Practical Applications of Mechanical Ventilation

Shaila Shodhan Kamat

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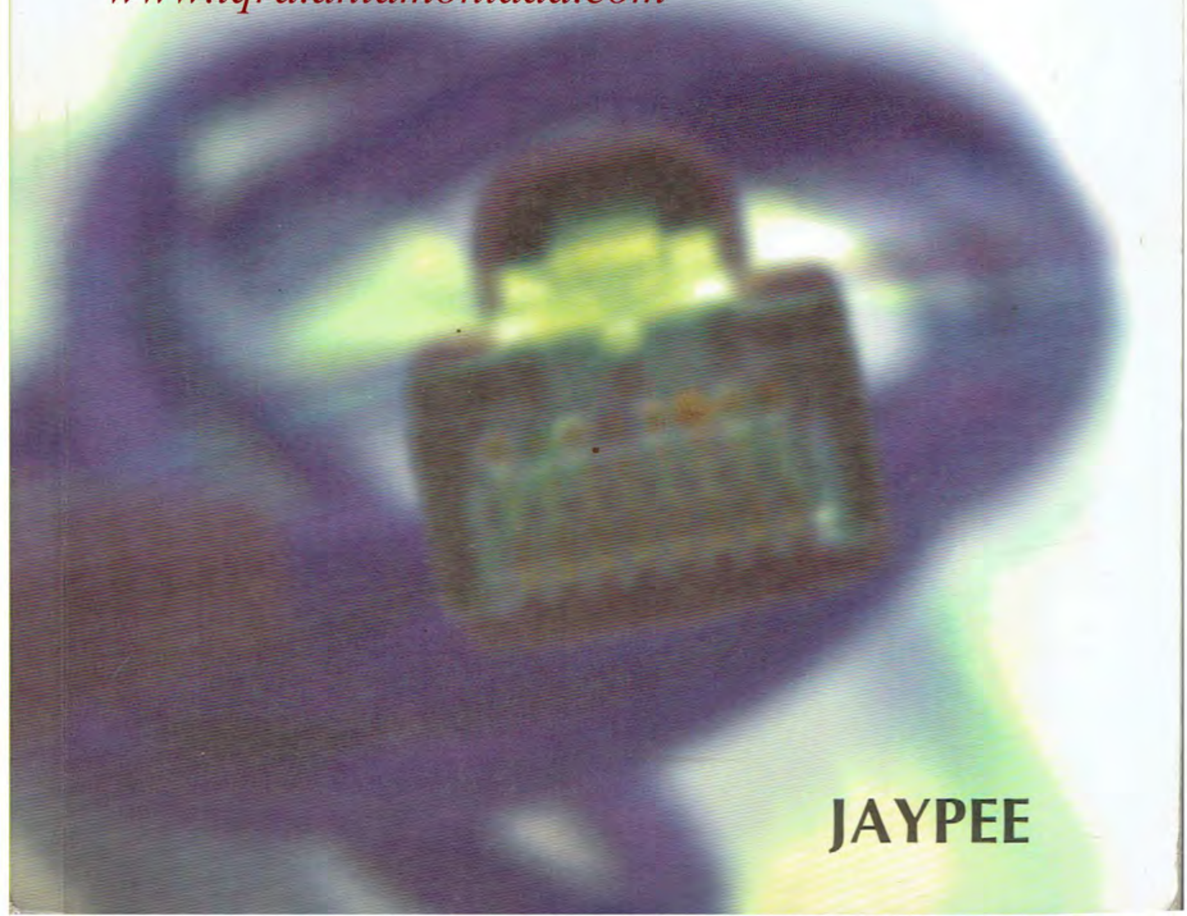

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Practical Applications of Mechanical Ventilation

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To
My doting grandparents
Late Mr Narcinva Damodar Naik
and
Late Mrs Laxmibai Narcinva Naik
Who truly understood the value of
Girls' education in the early 19th century
and
Who always loved me more than anybody in my life...

My loving parents
Late Mr Vassudeva N Naik
and
Mrs Rekha V Naik
For their unconditional love...

My adoring students
Who are my inspiration and strength...
and
All my patients
Whose unseen blessings helped me to complete this book...

FOREWORD

The recent advances in the field of mechanical ventilation have revolutionized the care of critically-ill needing artificial respiration. In addition to its role in intensive care, mechanical ventilation forms an integral part of management of most patients who receive general anaesthesia involving endotracheal intubation. Thus, mechanical ventilation is an indispensable part not only of most anaesthetic care but also of intensive care management.

To be able to cater to the individual needs of patients with different illnesses and to provide controlled ventilation in the operating rooms, it is mandatory to have an in-depth knowledge of the mechanical ventilation. To this end, Dr Shaila Shodhan Kamat has put in tremendous efforts to create this manual on *Practical Applications of Mechanical Ventilation*. I was delighted to go through the book, which is targeted at postgraduate students and fellows of anaesthesia and intensive care. I am happy to say that the language used is lucid and easily understood. I admire the zeal, enthusiasm and meticulous efforts Dr Shaila has taken for this endeavour and I feel privileged in having this opportunity to read the text.

I recommend this book to be read not only by postgraduate students of anaesthesia but also fellows, residents, teachers and faculty of intensive care medicine. I wish good luck to Dr Shaila in this venture and in future ventures too!

"I expect to pass through life but once. Therefore, if there be any kindness I can show, or any good thing I can do to any fellow being, let me do it now, and not defer or neglect it, as I shall not pass this way again".

– William Penn

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Director (Academics)
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FOREWORD

Teaching is an ancient activity; it requires a predisposition and ability to transmit one's own knowledge to others. It is also an innate quality that tends to strengthen over time due to the interaction between teacher and pupil that develops and intensifies during their association, and to the ready availability of constantly improving teaching methods.

It gives me immense pleasure to write a foreword for the book on *Practical Applications of Mechanical Ventilation* by Dr Shaila Shodhan Kamat. The various chapters have been so chosen as to cover the important topics of the curriculum of the postgraduate students. This book is going to help the practicing consultants as well.

Each chapter has been planned to be self-contained with cross referencing between chapters. The manual is produced as a comprehensive handbook. It is not intended to be a reference book, although topics are covered fairly extensively and sufficiently for most clinical situations.

I congratulate Dr Shaila for her endeavour in fulfilling the long felt need of such a book. I am more than sure that the readers will feel happy with the given information.

Dr Pramila Bajaj
Editor, Indian Journal of Anaesthesia
Senior Professor and HOD of Anaesthesiology
Department of Anaesthesiology
Additional Principal
RNT Medical College
Udaipur (Rajasthan)
25, Polo Ground, Udaipur - 313 001
Rajasthan, India

PREFACE

"It is far better to cure at the beginning than at the end."

Over the years, I have been involved in teaching which is my passion and love. The idea of this book came to my mind when I started taking regular lectures for my **PG** students and other practitioners. There were constant complaints from my students that there was no basic text available on ventilators which was clear, concise and easy to understand and which gave an overview of the physiologic basis of ventilation and ventilatory strategy for different diseases requiring intensive care.

The aim of this book is to meet these demands. It is an attempt to fill the gap and supplement rather than replace the many excellent textbooks already available, thereby allowing students to gain a foothold in the understanding of intensive care units and ventilators. This book is both, a theoretical as well as a practical guide for beginners. It aims at answering all the questions that arise during the daily work of postgraduate students in the daily routine of mechanical ventilation. This book has been written keeping in mind mainly young resident doctors of anaesthesiology, surgery and medicine confronting mechanical ventilation in the intensive care unit for the first time.

For certain reasons no references are quoted in the book. Firstly, it was never my objective to produce a reference source book. Secondly, the book is intended to be useful in day-to-day practice and it has been my experience that including references will make it difficult to achieve a concise format.

The attempt has been to produce a clear and practical understanding of ventilators and hence the book provides concise and accurate information on the basics of respiratory physiology, its clinical applications providing optimal knowledge in ventilatory strategy in intensive care. The book comprises of 43 chapters grouped into six parts to cover various aspects of ventilatory management. The aim of this book is to augment clinical teaching and inspire a more detailed study. The book has been written as a practical guide for the people working in an intensive care unit and postgraduate students. I felt the need for a detailed but simplified approach to cover the practical applications of mechanical ventilation.

I would be pleased and the effort would be worthwhile if readers find the book useful as a concise, up-to-date guide on the use of ventilators.

"Life's precious moments and knowledge do not have value unless they are shared."

Shaila Shodhan Kamat

ACKNOWLEDGEMENTS

"Your worst days are never so bad that you are beyond the reach of God's grace and your best days are never so good that you are beyond the need of God's grace"

Many of us may be lucky enough to have good family support but very few are blessed to have loving teachers. I am one of those fortunate ones. The way my family, my mother, my siblings and my uncles and aunts have supported me, the same way I received strong support from my teachers, whom I have adored not just at a tender age but even today when I have crossed the half century mark of my life. I really must have done some good deeds to have had so many wonderful teachers who have blessed me from their soul to help to be what I am today and in turn write this book on *Practical Applications of Mechanical Ventilation.* I am fortunate to have valuable suggestions and the foreword written by *Dr Muralidhar K and Dr Pramila Bajaj.*

I am extremely thankful to my little sister *Rukma Naik* for improving my text and to *Dr Marilyn Nazareth* (Professor and HOD), Department of Anaesthesiology for supporting me in my academic growth.

The book would not have been completed without the support of my friends and well wishers, who are too numerous to mention individually. I am very thankful to *all my students* (present and past) whose love, inspiration and wholehearted admiration for my teaching has always been a great strength to me for my continuous academic and spiritual growth.

I would like to mention special thanks to my mother *Mrs. Rekha V Naik* for her constant, unconditional support and love, my uncles late *Mr Anant (Babu) N Naik* and *Mr Damodar N Naik* for their love and protection which they have showered on me throughout my life.

Behind every successful woman there is the helping hand of her husband, children and siblings. I wholeheartedly appreciate the support and patience of my husband *Mr Shodhan,* my daughters *Salonee* and *Asmani* and my loving sister *Nita Manerkar.*

My sincere thanks to *Shri Jitendar P Vij (Chairman and Managing Director), Mr Tarun Duneja* (Director-Publishing) of M/s Jaypee Brothers Medical Publishers (P) Ltd, New Delhi and his team for publishing this book.

Though I am not an overtly religious person, I have always felt God's presence in my life and without his kind hand, this book wouldn't be possible.

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PLATE 1

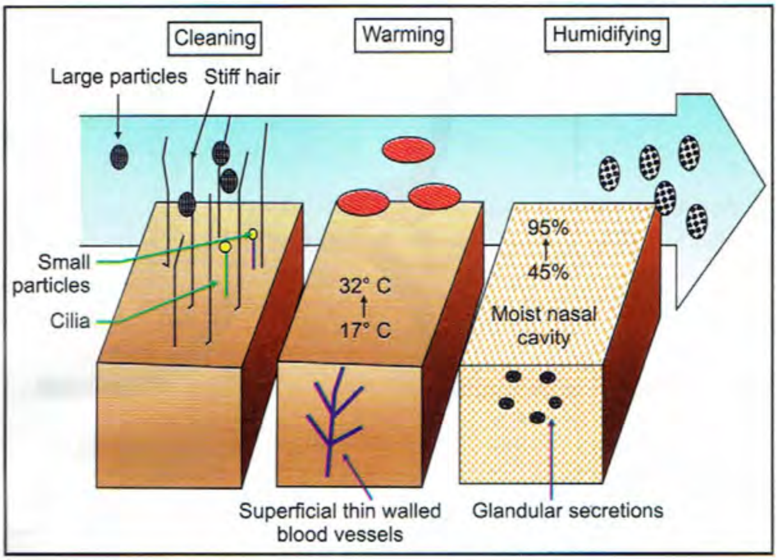


Figure 1.1: Functions of nasal cavity

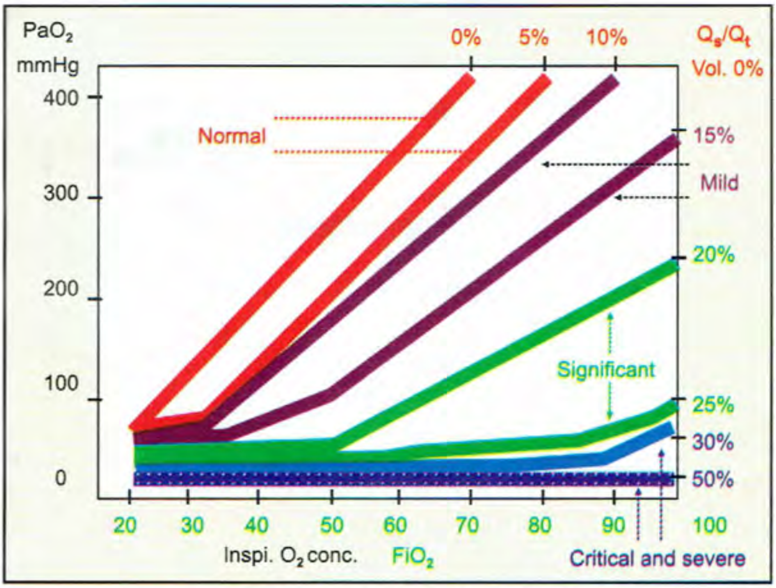


Figure 7.3: Analysis of iso-shunt-diagram

PLATE 2

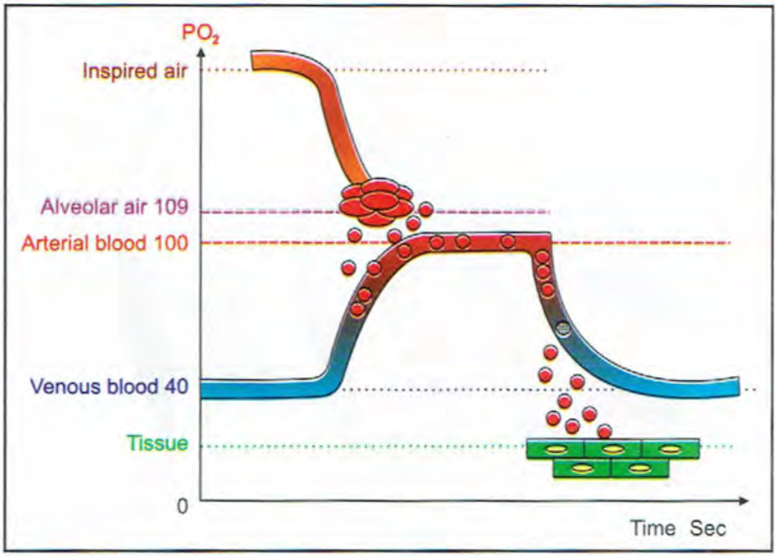


Figure 9.3: Diffusion of oxygen over alveolar membrane and at tissue level

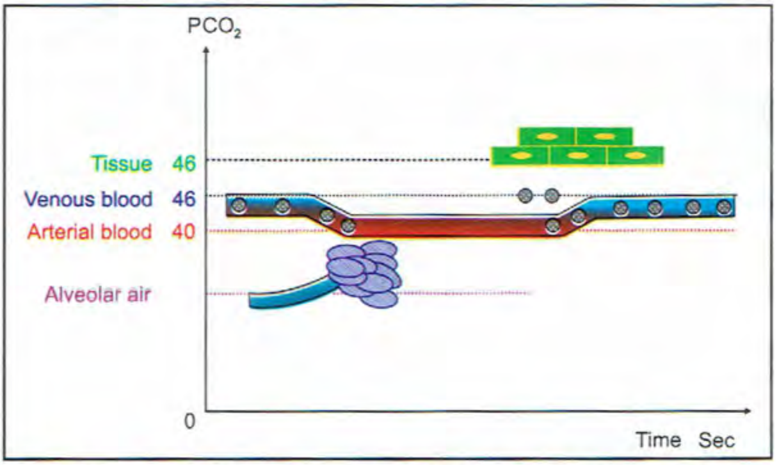


Figure 9.4: Diffusion of carbon dioxide over alveolar membrane and at tissue level

PLATE 3

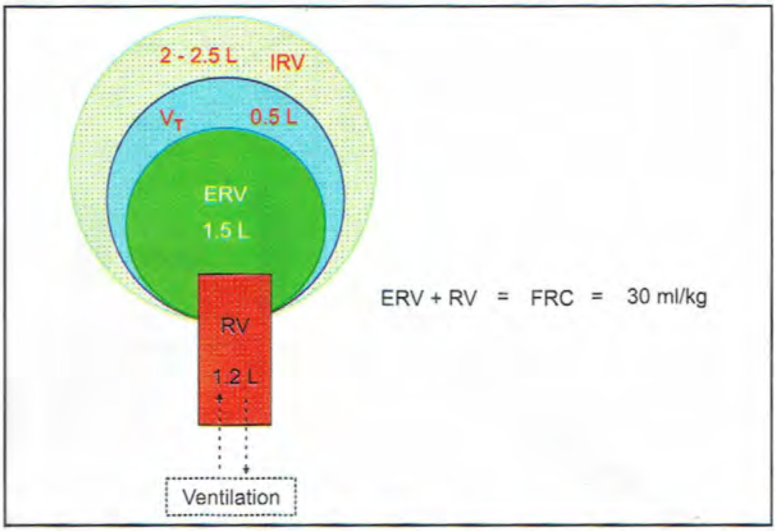


Figure 10.3: Static lung volumes and capacities

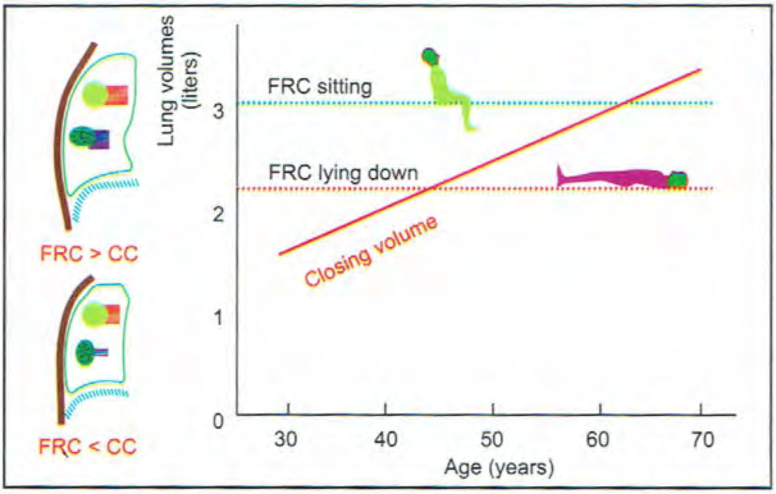


Figure 10.8: Closing volume and FRC in relation to age and body position

PLATE 4

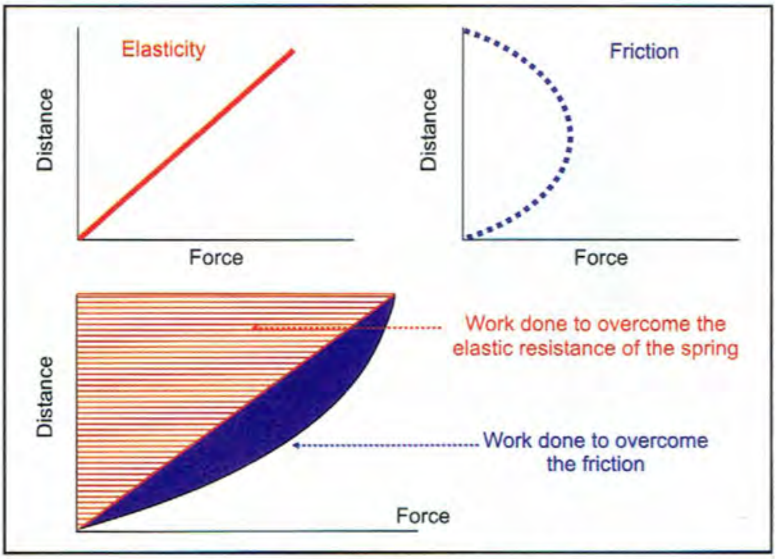

Elasticity
Distance
Force
Friction
Distance
Force
Distance
Work done to overcome the elastic resistance of the spring
Work done to overcome the friction
Force

Figure 11.5: Total work required to move the block

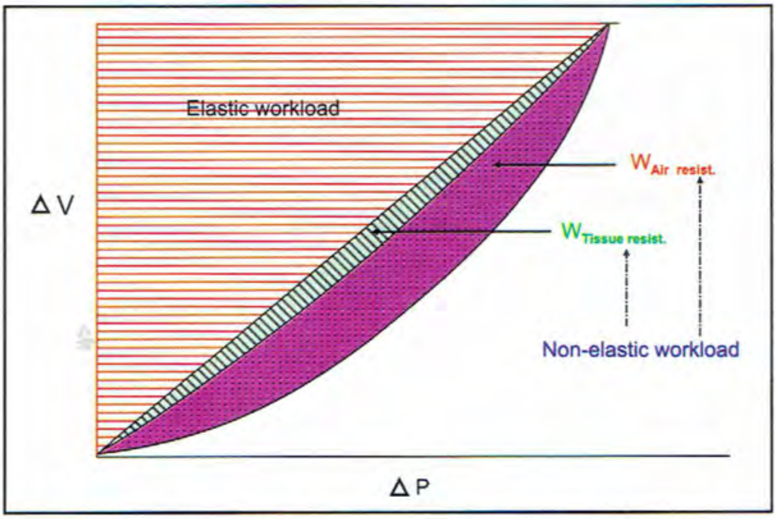


Figure 11.9: Work done during inspiration

PLATE 5

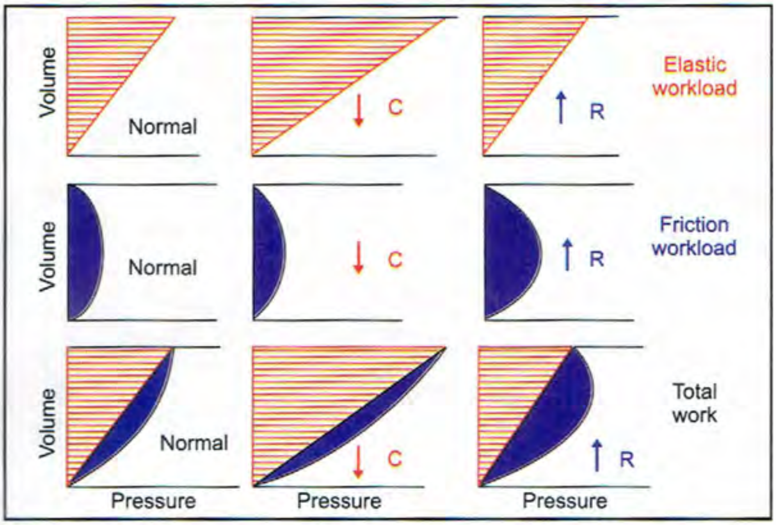


Figure 11.11: The effects of decreasing lung compliance and increasing airway resistance on WOB

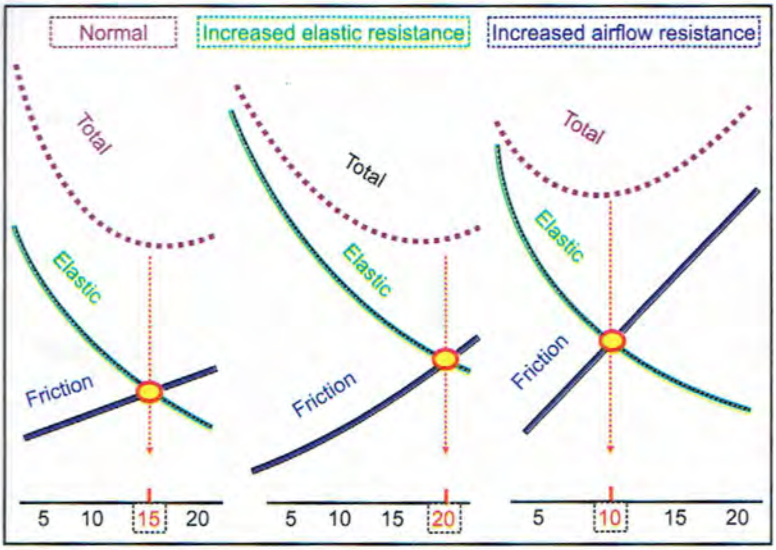


Figure 11.16: Comparison of normal, increased elastic and obstructive work load

PLATE 6

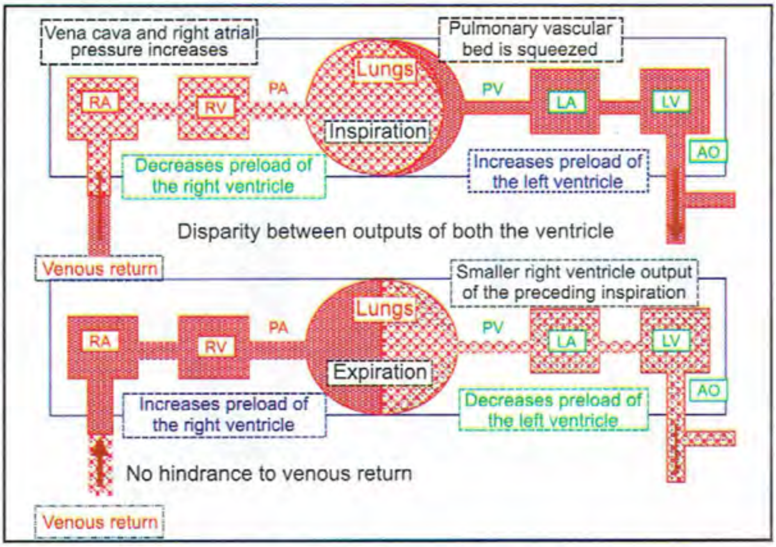

Vena cava and right atrial pressure increases
Pulmonary vascular bed is squeezed
RA
RV
PA
Lungs
Inspiration
PV
LA
LV
AO
Decreases preload of the right ventricle
Increases preload of the left ventricle
Disparity between outputs of both the ventricle
Venous return
Smaller right ventricle output of the preceding inspiration
RA
RV
PA
Lungs
Expiration
PV
LA
LV
AO
Increases preload of the right ventricle
Decreases preload of the left ventricle
No hindrance to venous return
Venous return

Figure 13.4: Circulation showing the effects of positive pressure breath on the preloads of the right and left ventricle

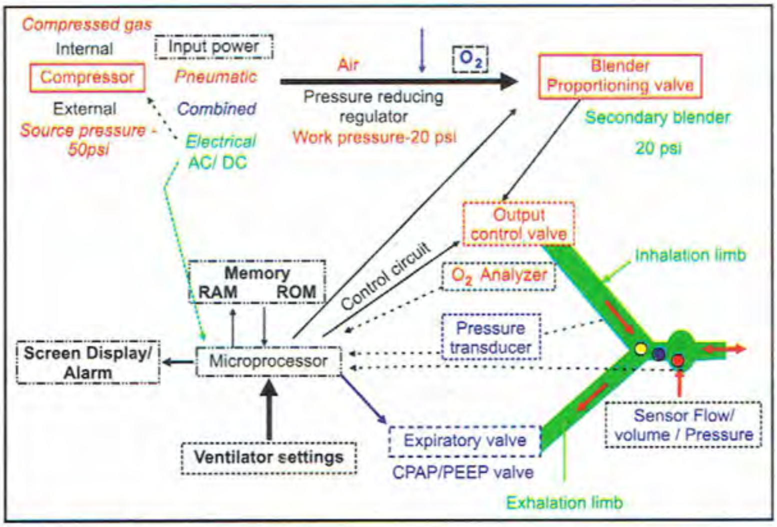


Figure 15.11: Diagrammatic representation showing the operation of a microprocessor controlled ventilator

PLATE 7

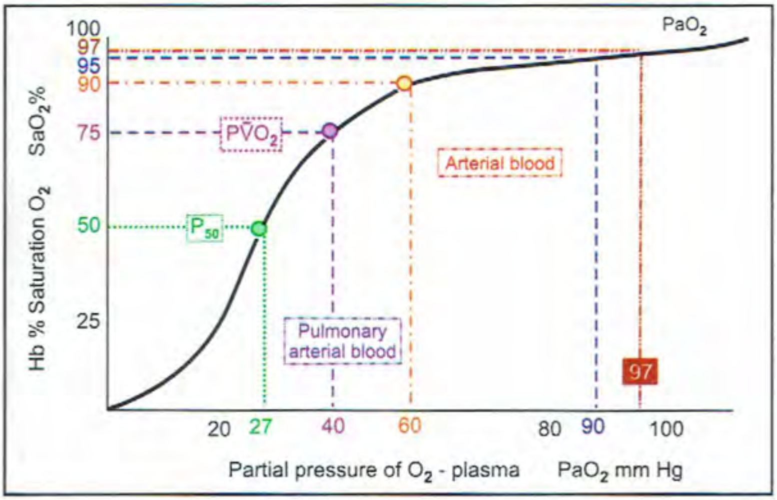


Figure 16.3: Oxygen dissociation curves

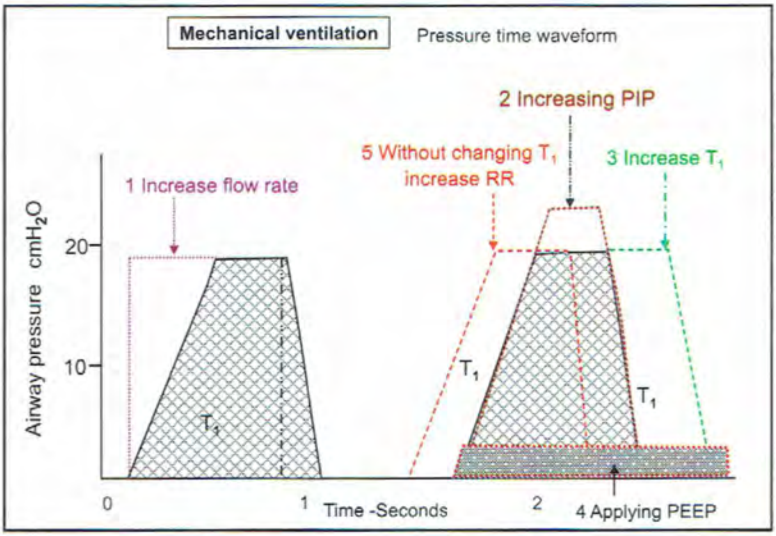


Figure 16.6: Ways to increase MAP

PLATE 8

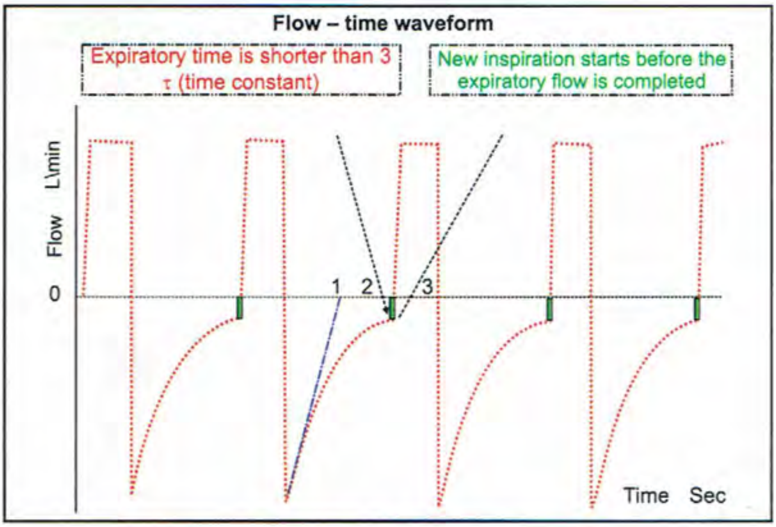


Figure 18.4: Auto-PEEP

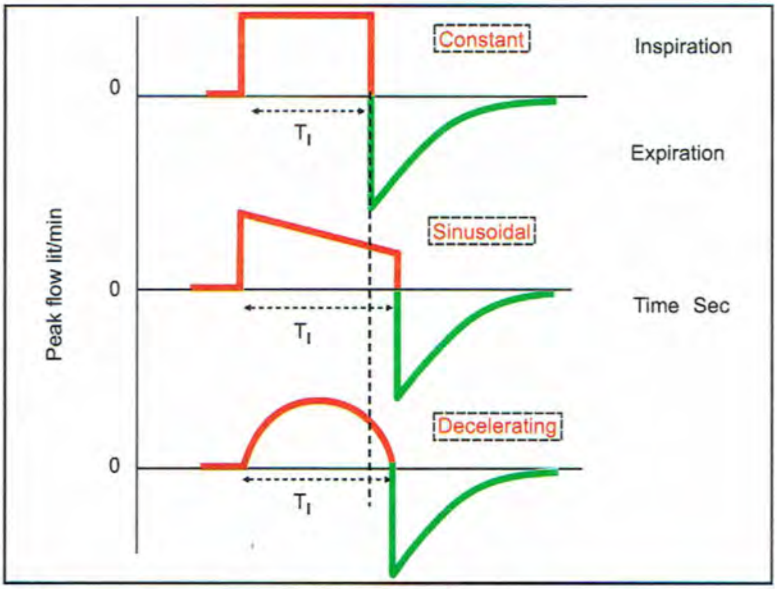


Figure 18.8: Comparisons of duration of inspiratory time for different inspiratory flow pattern

PLATE 9

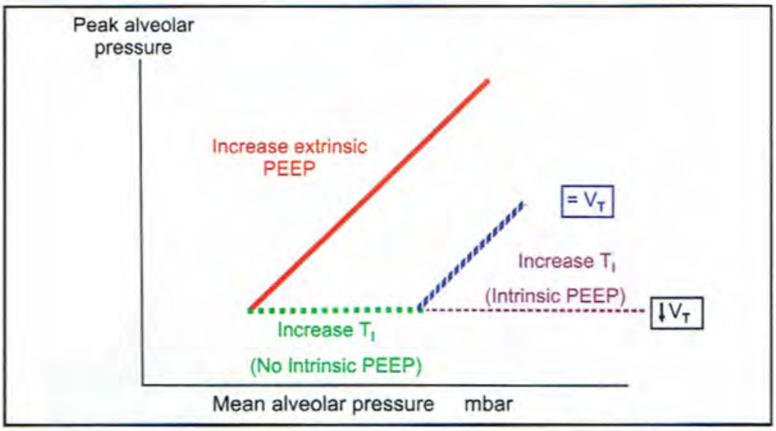


Figure 20.1: Relationship of peak alveolar pressure to MAP using various strategies to increase mean alveolar pressure

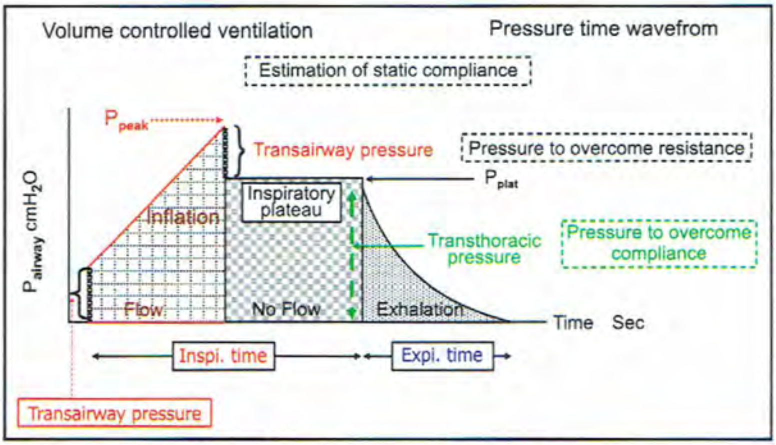


Figure 20.7: Volume-controlled ventilation

PLATE 10

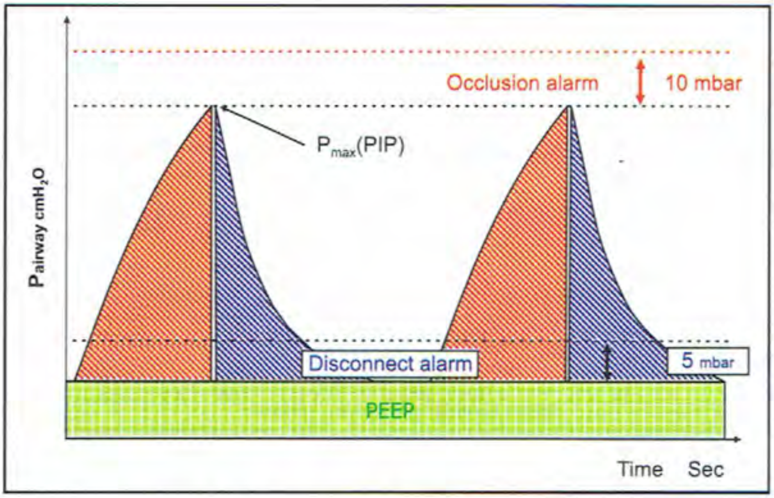


Figure 22.1: Alarm limits for ventilation pressure

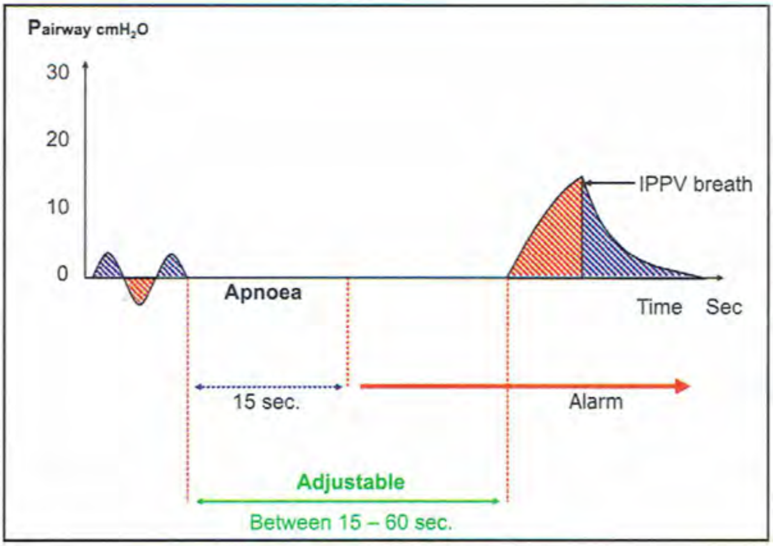


Figure 22.2: Apnoea ventilation alarm

PLATE 11

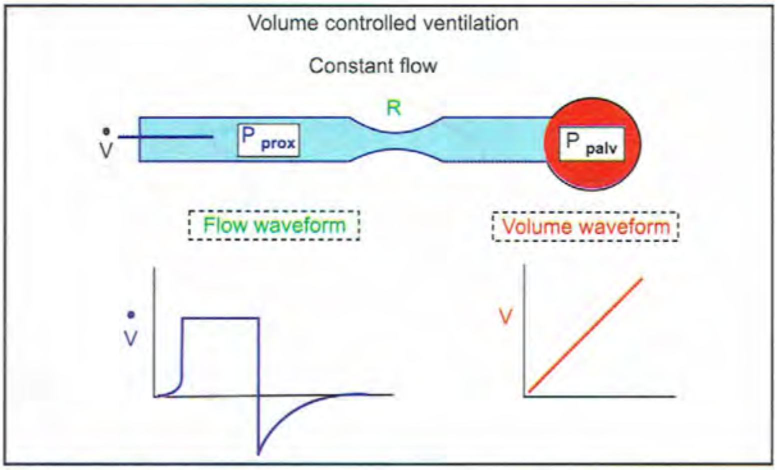


Figure 25.2: Flow/Volume waveform produced by constant flow

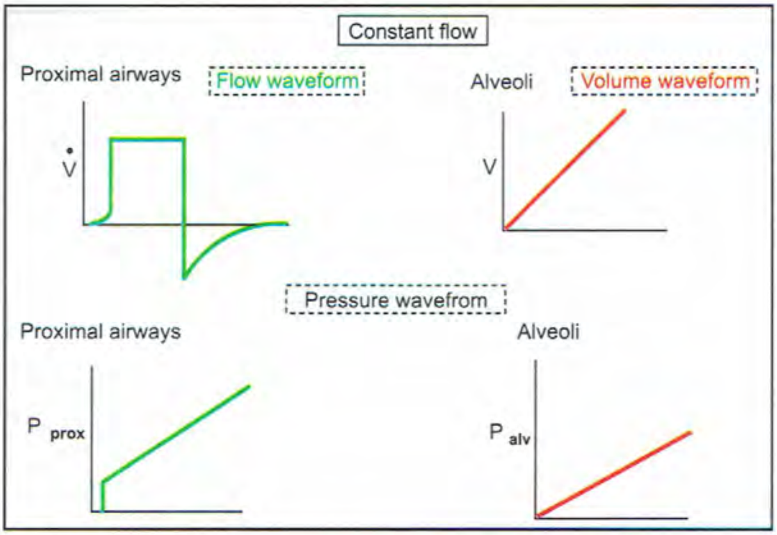


Figure 25.3: Flow/Pressure waveform produced by constant flow

PLATE 12

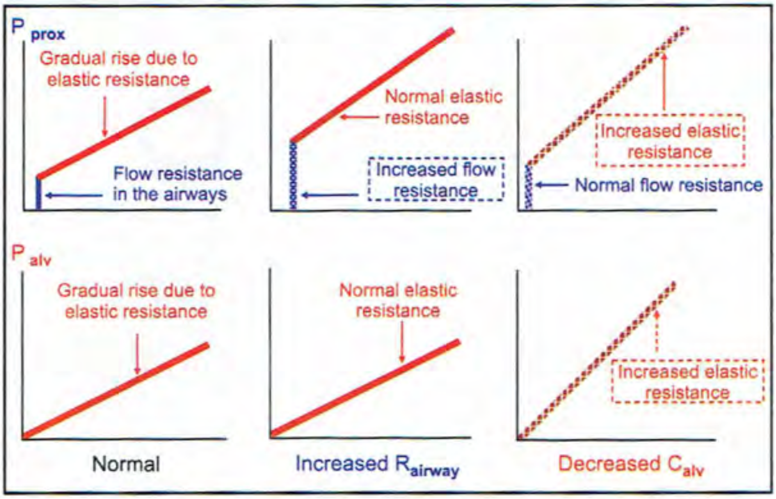


Figure 25.4: Flow/ Pressure waveforms produced by constant flow in normal and abnormal lung

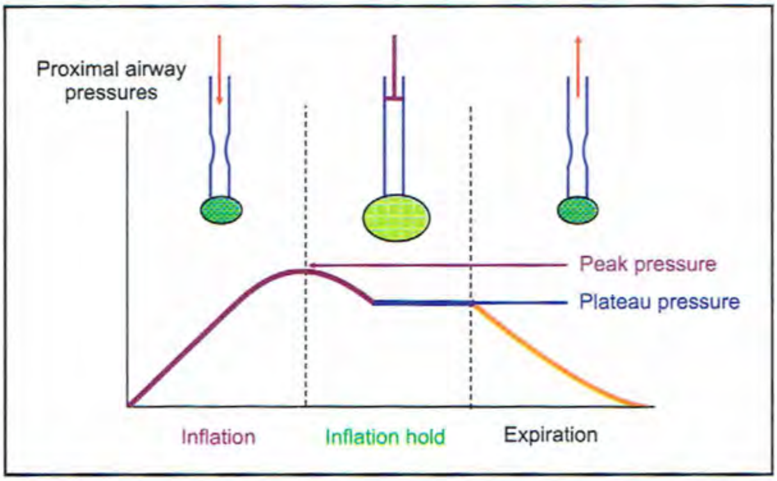


Figure 25.7: Proximal airway pressures at the end of positive-pressure lung inflation

PLATE 13

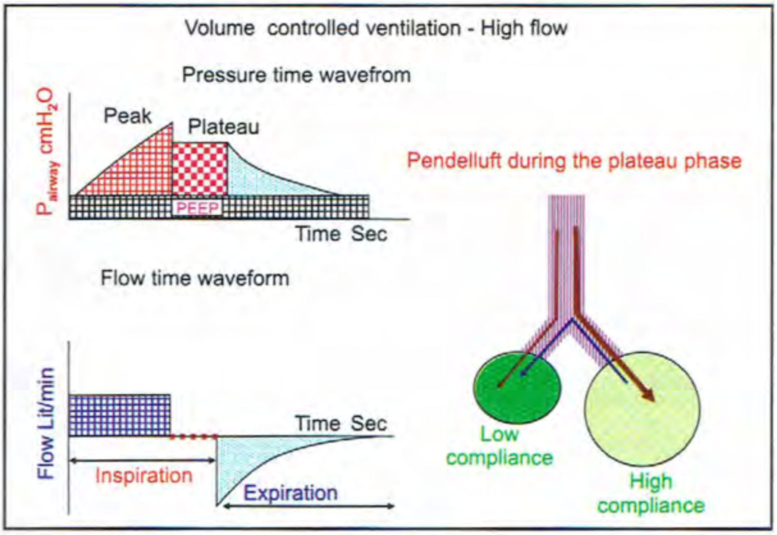


Figure 29.7: High inspiratory flow in volume controlled ventilation

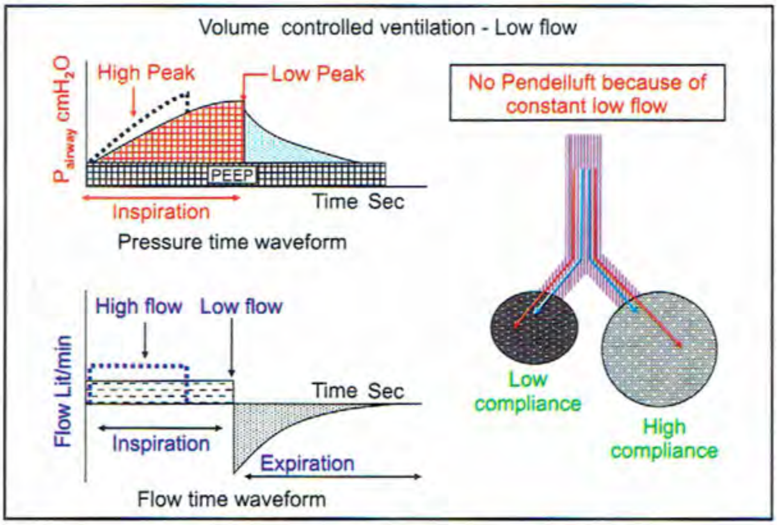


Figure 29.8: Low inspiratory flow in volume controlled ventilation

PLATE 14

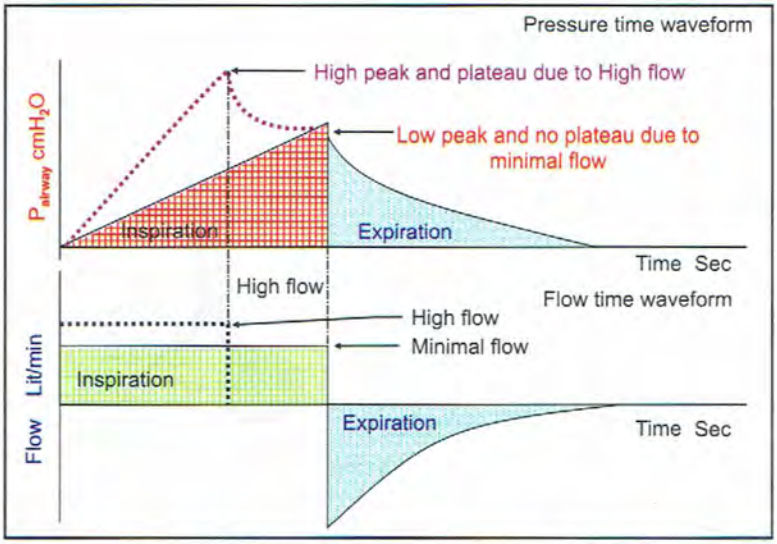


Figure 29.9: Minimal flow in volume controlled ventilation

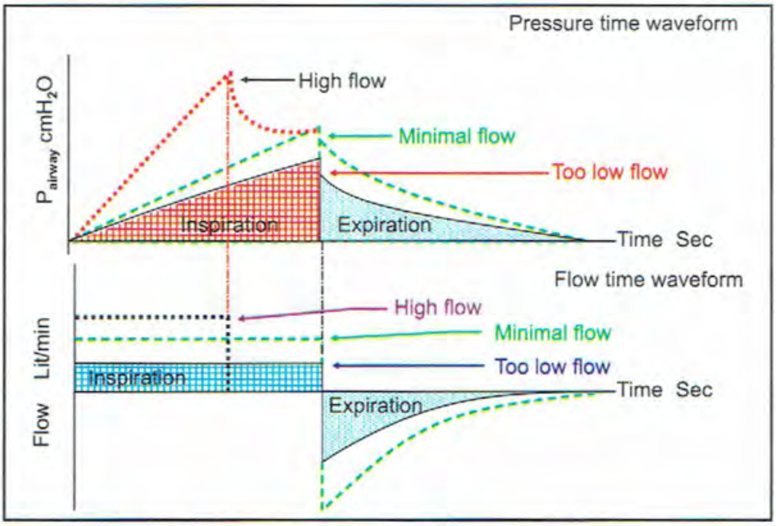


Figure 29.10: Too low flow in volume controlled ventilation

PLATE 15

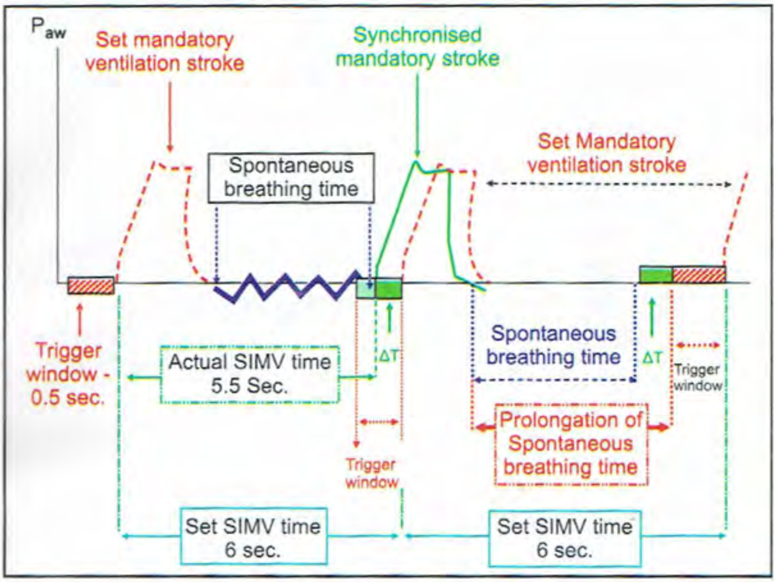

P$_{aw}$
Set mandatory ventilation stroke
Synchronised mandatory stroke
Spontaneous breathing time
Set Mandatory ventilation stroke
Trigger window - 0.5 sec.
Actual SIMV time 5.5 Sec.
ΔT
Spontaneous breathing time
ΔT
Trigger window
Prolongation of Spontaneous breathing time
Trigger window
Set SIMV time 6 sec.
Set SIMV time 6 sec.

Figure 31.7: Mechanism of synchronization

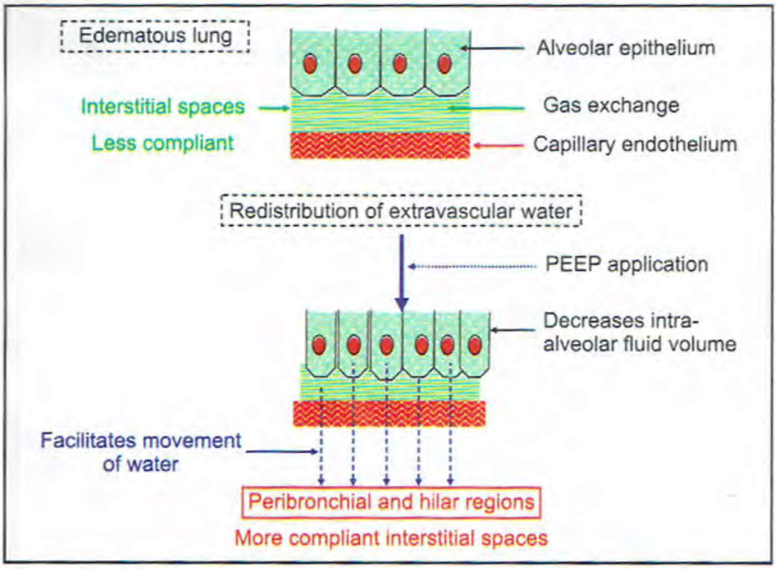

Edematous lung
Alveolar epithelium
Interstitial spaces
Gas exchange
Less compliant
Capillary endothelium
Redistribution of extravascular water
PEEP application
Decreases intra-alveolar fluid volume
Facilitates movement of water
Peribronchial and hilar regions
More compliant interstitial spaces

Figure 33.6: Re-distribution of extravascular water

PLATE 16

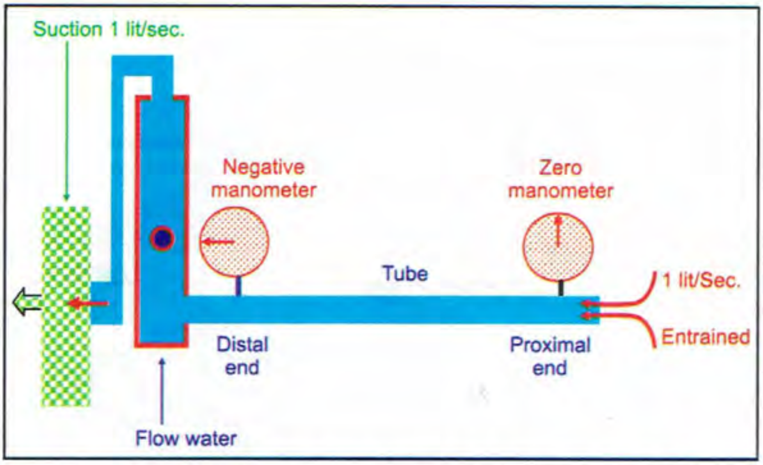
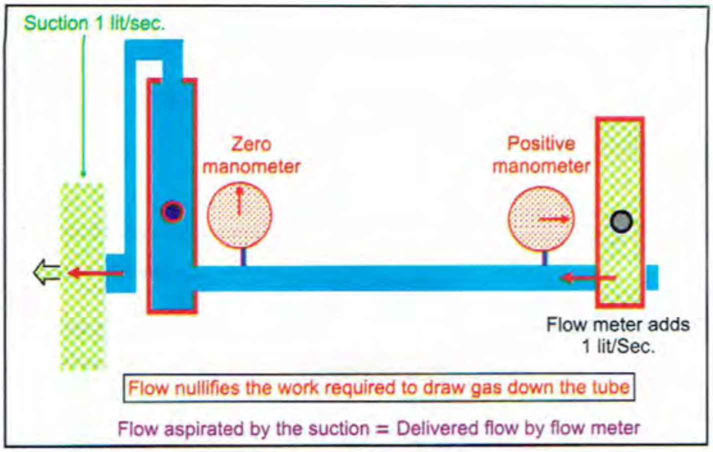
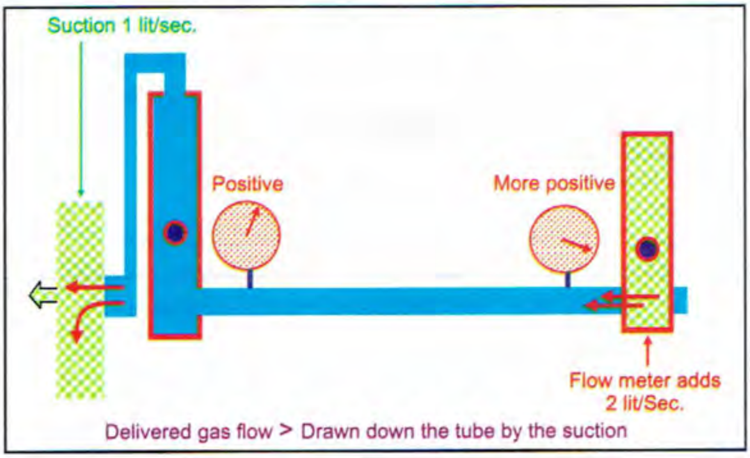


Figure 34.1: Work to be done to draw the gas down the tube



Suction 1 lit/sec.
Zero manometer
Positive manometer
Flow meter adds 1 lit/Sec.
Flow nullifies the work required to draw gas down the tube
Flow aspirated by the suction = Delivered flow by flow meter

Figure 34.2: Flow nullifies the work required to draw gas down the tube



Suction 1 lit/sec.
Positive
More positive
Flow meter adds 2 lit/Sec.
Delivered gas flow > Drawn down the tube by the suction

Figure 34.3: Attenuates elastic workload

PART-I

Respiratory Physiology

CHAPTER

1

Anatomy of Respiration

Respiration is the uptake of oxygen by the body and the elimination of carbon dioxide. It can be divided into:

- *External respiration:* Ventilation and gas exchange is called external respiration.
- *Internal respiration:* Combustion or biologic oxidation of nutrients by oxygen, to carbon dioxide and water at cellular level is called internal respiration.

RESPIRATORY ORGANS

The respiratory organs can be divided into:

1. Upper airway
    - Nasal cavity
    - Oral cavity
    - Pharynx
2. Lower airway
    - Larynx
    - Trachea
    - Bronchial tree
3. Lungs.

Nasal Cavity (Fig. 1.1)

The nasal cavity has important functions (anesthetic significance):-

1. *Breathing through the nose:*

   The adult patient breathes through the nose unless there is some form of an obstruction such as a nasal polyp. In normal subjects the

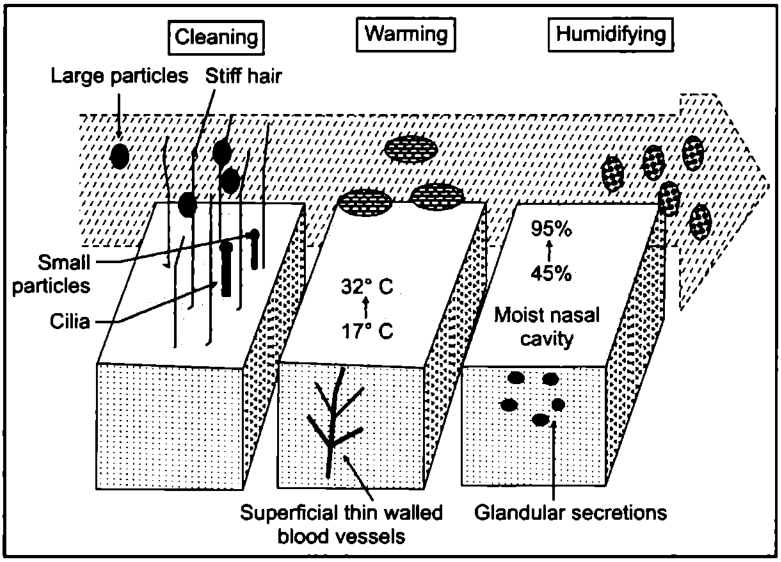


Figure 1.1: Functions of nasal cavity (For colour version *see* Plate 1)

resistance created by the nasal passage is one and a half times greater than in mouth breathing. Deflection of the nasal septum may diminish the size of the nasal passage, reducing the size of the nasal endotracheal tube and increasing the airway resistance.

2. *Cleaning:*
   Stiff hair, spongy mucous membrane and ciliated epithelium comprise a powerful defence against any organism. The hair present inside the nose nearest to the nostrils, clears the air of larger particles. The cilia are responsible for trapping and removing small foreign particles.
3. *Warming the inhaled air:*
   The vascularity of mucosa helps to maintain a constant temperature. In the nasal cavity, there are a number of superficial, thin walled blood vessels which radiate heat and thereby warm the inspired air from 17°C to 37°C when it is passing through the nasal passage.
4. *Humidification of the inhaled air:*
   The nasal cavity is kept moist by glandular secretions which also humidify the air. Relative humidity of air is 45-55% but the bronchi and alveoli require 95% for adequate functioning. The inspired air, which passes through the nose, is thus fully humidified.

   Anaesthetic Significance of Humidification

   - During treatment on a ventilator the importance of correct humidification and warming of the inspired gas has to be considered, as the gas is supplied through an endotracheal tube and not through the nose.

- If the inhaled air does not pass through the nose, (for example when breathing through the mouth) partial drying of the mucous membranes of the lower airways occurs, making them more prone to infection.

Larynx

The larynx protects the lower airway by closing the glottis (for example during swallowing). The extrapulmonary airway (larynx) is at its narrowest at the vocal cords in an adult and at the level of the cricoids in children. Any further narrowing at the vocal cords can give rise to considerable respiratory distress. The laryngeal mucosa can become oedematous due to anaphylactic reactions or postextubation. This can cause life-threatening problems.

Trachea

The trachea is a cartilaginous tube made up of 16-20 horseshoe shaped cartilage rings which are incomplete posteriorly. The trachea measures about 10-12 cm in length and 11-12.5 mm in diameter in an adult.

Anaesthetic Significance

The trachea moves during respiration and with a change in position of the head. On deep inspiration the carina can descend as much as 2.5 cm and the extension of the head can increase the length of the trachea by 25-30%. Therefore always check the position of the endotracheal tube for accidental extubation or endobronchial intubation after any change in the position of the head.

Bronchial Tree (Figs 1.2 and 1.3)

The bronchial tree subdivides into 23 generations, the 23rd generation being alveoli. The total diameter of the airways increases considerably towards the periphery. The bronchioles begin in the 10th generation and their diameter measures less than 1 mm, the walls are free of cartilage, rich in smooth muscle fibres and the epithelium no more contains mucous producing cells. Upto the 16th generation the bronchi play no role in gas exchange, their only purpose is the transportation of air. The gas exchange zone begins with the respiratory bronchioles where the smooth muscle fibres become rarer and there is an increase in alveolar budding.

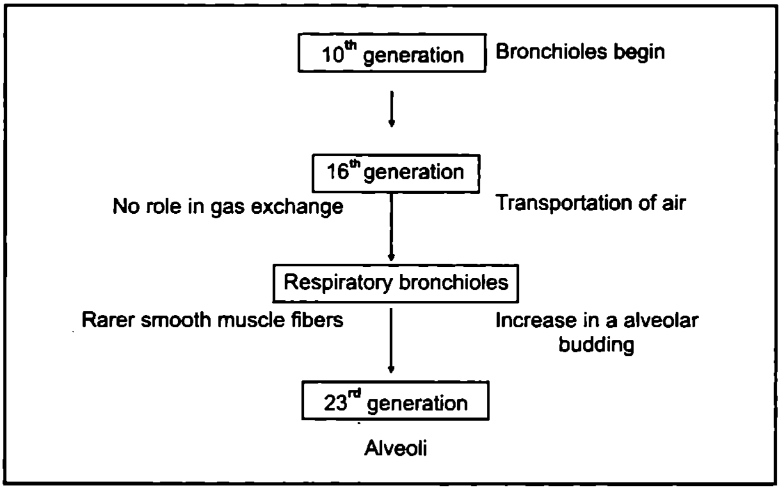


Figure 1.2: Subdivisions of bronchial tree

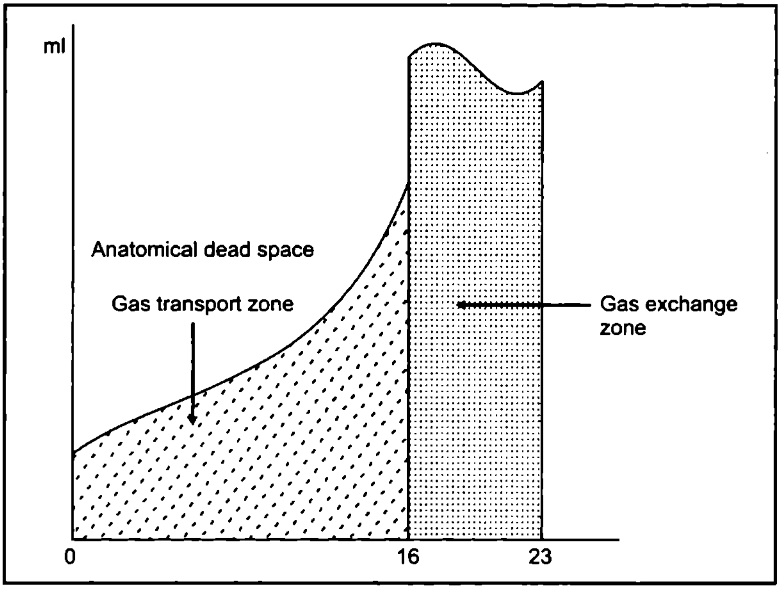


Figure 1.3: Generations of airways

Right Main Bronchus (Fig. 1.4)

The right main bronchus is wider and shorter than the left, being only 2.5 cm long. The angulations of both bronchi are not equal and it is 25° for right bronchus. In small children, under the age of three years, the angulations of the two main bronchi at the carina are equal on both sides.

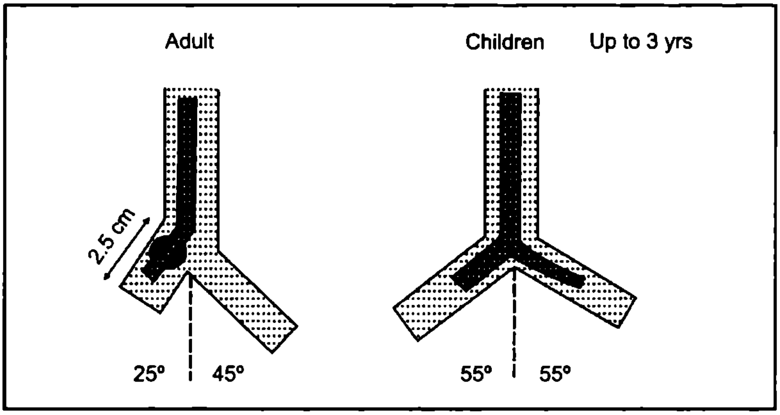


Figure 1.4: Angle of the main bronchi

Clinical Applications

Adults

- Greater tendency for right endobronchial intubation
  In adults the right bronchus is more vertical than the left main bronchus and hence there is a greater tendency for either endotracheal tubes or suction catheters to enter this lumen.
- Blocking bevel end of the tube
  In the event of an endotracheal tube being inserted too far, the bevelled end of the tube may get blocked off because of it lying against the mucosa on the medial wall of the main bronchus.
- Difficult to occlude
  The short length of the right bronchus also makes the lumen difficult to occlude when this is required for thoracic anesthesia.

Children under the age of three years: Due to equal angulations of the two main bronchi at the carina, endotracheal tubes or suction catheters can enter either lumen.

MUCOCILIARY CLEARANCE

It is the most important cleansing mechanism of the peripheral airways. Throughout the respiratory tract, the continuous activity of the cilia is probably the single most important factor in the prevention of accumulation of secretions.

In the nose the material is swept towards the pharynx whereas in the bronchial tree the flow is towards the entrance to the larynx. The coordinated movement of numerous cilia is capable of moving large quantities of material but their activity is greatly assisted by the mucous covering.

Mucous Layers

The mucous layer covering the cilia consists of two layers:

- Superficial gel layer (Fig. 1.5).

  An outer layer of thick, viscous mucous is designated to entrap dust and micro-organisms. With each beat, the tips of the cilia just come in contact with the outer layer. Acting in unison, they set the outer mucous layer in motion and with gathering momentum this flows towards the pharynx and larynx. The cilia cannot work without this blanket of mucous.

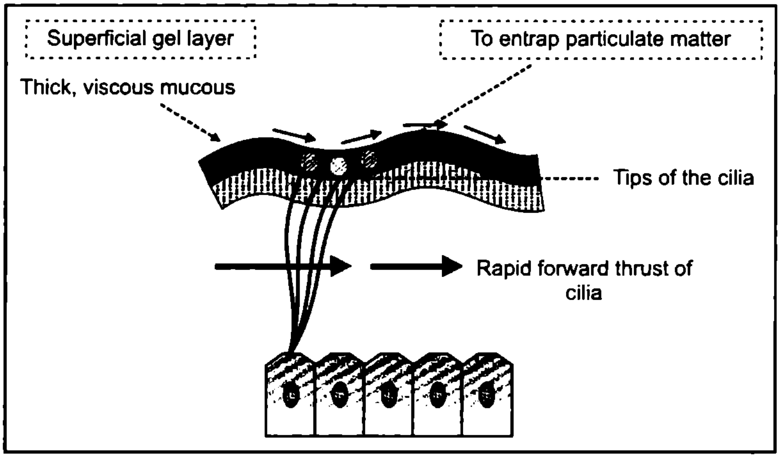


Figure 1.5: Superficial gel layer

- Fluid sol layer (pericilary fluid layer) (Fig. 1.6)

  An inner layer, surrounding the cilia, is of thin, serous fluid that is required to lubricate the action of the ciliary mechanism. Ciliary movement consists of a rapid forward thrust followed by slow recoil which occupies about four-fifths of the cycle. Their action can be compared to that of a belt system of the platform on which the bags rest. The platform corresponds to the blanket of mucous and the propulsive force of the belt is represented by the action of the cilia.

Visco-mechanical Dissociation

Visco-mechanical dissociation occurs when: (Fig. 1.7)

- The periciliary fluid layer is too deep e.g. pulmonary oedema, overdose of mucolytics etc.
- The periciliary fluid layer is too shallow e.g. dehydration, insufficient moistening of the administered gases during mechanical ventilation. When there is insufficient moisture within the airways the transport function of the respiratory cilia stops rapidly.

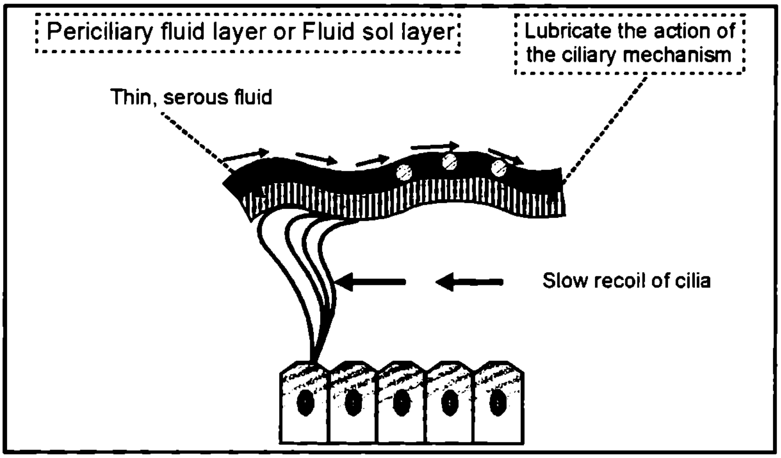

Periciliary fluid layer or Fluid sol layer
Lubricate the action of the ciliary mechanism
Thin, serous fluid
Slow recoil of cilia

Figure 1.6: Inner fluid sol layer

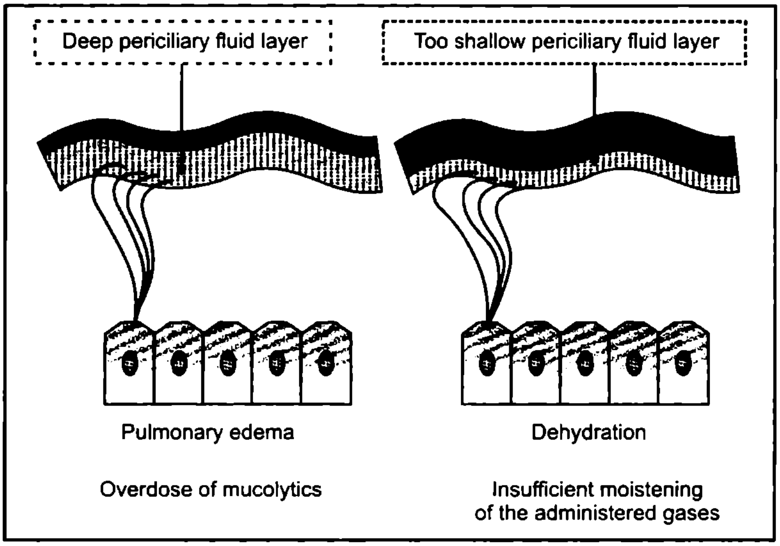

Deep periciliary fluid layer
Too shallow periciliary fluid layer
Pulmonary edema
Overdose of mucolytics
Dehydration
Insufficient moistening of the administered gases

Figure 1.7: Visco-mechanical dissociation

Factors Affecting Mucociliary Clearance

1. Toxic gases

   Toxic gases (NO_2, SO_2) and tobacco smoke have the same effect, depressing ciliary activity.

2. Drugs used in anesthesia

   Anesthetic agents (thiopentone) and other drugs such as atropine or beta blockers also reduce the mucociliary clearance.

   - Anticholinergic drugs

     Anticholinergic drugs with dry anesthetic gases produce dry mucosa. This produces an inflammatory reaction producing excessive mucous giving rise to tracheitis, pulmonary collapse and bronchitis.

- Volatile general anesthetics
  A volatile general anesthetic not only slows the propelling mechanism but also limits the production of suitable mucous.
- Beta stimulation
  Beta adrenergic substances, sympathetic stimulation and theophyllines stimulate mucociliary clearance.

3. Infections
   Infections attenuate the mucociliary clearance by way of a further ciliostatic effect.
4. Tussive clearance
   Mucociliary transportation is enhanced further by coughing (tussive clearance) after increasing the pressure by closing the glottis. The sudden opening of the glottis at maximum pressure leads to enormous local airflow in large airways enabling great masses of mucous to be removed suddenly.

Alveoli

The alveoli are composed of the alveolar epithelium, the epithelial basement membrane and the capillary endothelium. The total of all these layers is referred to as alveolar capillary membrane. It measures 1μ in thickness, thus representing a short distance for gas exchange between the alveolar space and the capillary space. Oxygen moves from the inspired air to the deoxygenated venous blood. Carbon dioxide moves in the opposite direction from the venous blood to the air in the lungs. This movement is carried out by passive diffusion, which means oxygen crosses passively through a membrane from a greater concentration in the lungs to a lower concentration in venous blood. Carbon dioxide crosses through the membrane in the opposite direction by the same principle.

The alveolar epithelium in alveolar ducts consists of flat epithelial cells or type I cells and alveolar granulocytes or type II cells, which produce surfactants. Foreign particles that gain access to the alveolar space are removed by alveolar macrophage by phagocytosis.

CONTROL OF VENTILATION

Regulation of gas exchange is possible because the level of ventilation is carefully controlled. Respiration is a largely involuntary process involving rhythmic impulses from the higher center of control of breathing in the brain which are passed on through efferent pathways to the muscles of respiration. There are two types of control:

- Voluntary control: This is initiated by the cerebral cortex.
- Involuntary or automatic rhythm control: This involves medulla, pons, limbic system (emotional response), hypothalamus (temperature regulation) and other subcortical structures.

There are three basic elements of the respiratory control system (Fig. 1.8)

- Sensor: The sensor gathers information and feeds it to the central controller.
- Central controller: The central controller, which is in the brain, coordinates the information from various sensors and in turn sends impulses, to the respiratory muscles.
- Effectors: Effectors are respiratory muscles which cause ventilation.

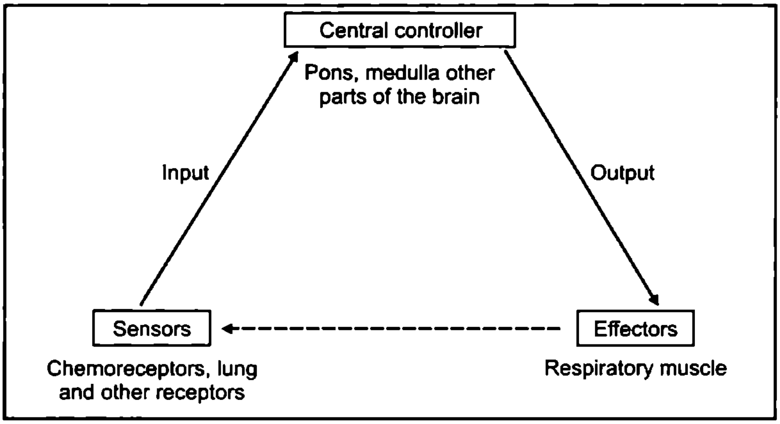

Central controller
Pons, medulla other parts of the brain
Input
Output
Sensors
Effectors
Chemoreceptors, lung and other receptors
Respiratory muscle

Figure 1.8 : Basic elements of the respiratory control system

By changing ventilation the respiratory muscles reduce the output of the sensors (negative feedback).

Brainstem

The normal automatic process of breathing originates in impulses that come from the brainstem. The cortex can override these centres if voluntary control is desired. Nerve cells which are situated in the pons and medulla are responsible for the automatic rhythm of breathing. These cells are arranged in functional groups known as the respiratory center.

The respiration is normally initiated and controlled by neural output from the respiratory centre.

The three main groups of neurons (Fig. 1.9) are:

- Medullary respiratory centre
- Apneustic centre
- Pneumotaxic centre

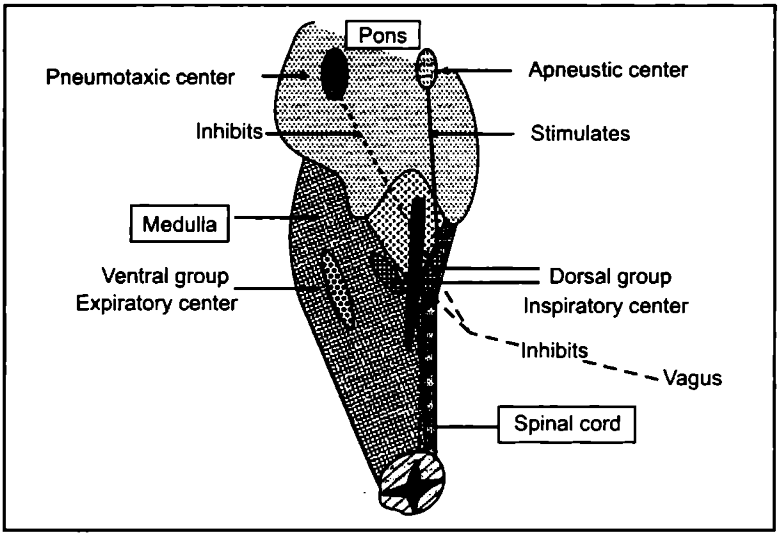


Figure 1.9: Respiratory centre or pacemaker of ventilation

1. *Medullary Respiratory Centre*

   The medullary centre is situated in the reticular formation beneath the caudal end of the floor of the 4th ventricle. The medullary centres have connections with the higher centres, the reticular activating system and the hypothalamus. The medullary center has been divided into two different parts:

- *Inspiratory centre*

  Dorsal group: Mainly inspiratory (I) neurons–lying more caudal and deep to the expiratory centre. Inspiratory neurons control the descending spinal cord pathways to the motor neurons innervating the muscles of inspiration. Vagal and glossopharyngeal nerves transmit signals from peripheral chemoreceptor to the inspiratory area. In addition, vagal nerves transmit sensory signals from the lung that help to control inflation and the rate of breathing.

- *Expiratory centre*

  Ventral group: Both inspiratory (I) and expiratory (E) neurons situated in the reticular substance under the floor of 4th ventricle.

Expiratory neurons control the descending spinal cord pathways to the motor neurons innervating the muscle of expiration (these are somatic motor nerves, not autonomic nerves).

Reciprocal Innervations for Respiratory Muscles (Fig. 1.10)

The inspiratory and expiratory neurons exhibit reciprocal innervations (mutually inhibitory) and are not generally active at the same time. The expiratory area is quiescent during normal quiet breathing because ventilation is then achieved by active contraction of the respiratory muscles (chiefly the diaphragm), followed by passive relaxation of the chest wall.

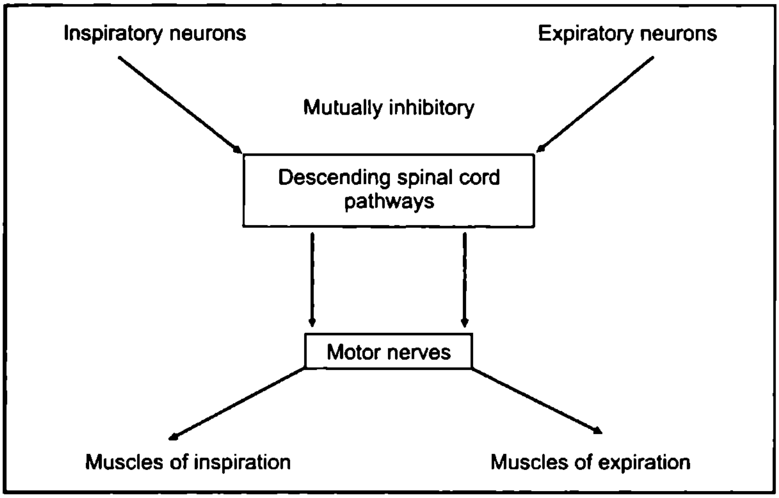


Figure 1.10: Reciprocal innervations for respiratory muscles

Possible organization of the respiratory centre (Fig. 1.11) are as follows.

2. *Apneustic Centre*

The apneustic center is situated in the lower two third of the pons and provides the initial stimulus which begins inspiratory activity in the inspiratory center in the medulla. The apneustic center also acts as a central station for vagal inhibitory impulses. The section of the brain at the junction of the upper one third and lower two thirds of the pons, separating the pneumotaxic center and the apneustic centre, leads to slower and deeper breathing.

If the vagus nerves on both sides are also divided, a state of inspiratory spasm appears due to uninhibited activity of the inspiratory center in the medulla, termed as apneusis. This is interrupted by expiratory gasps called apneustic breathing. The inference is that uninhibited action of the apneustic center causes prolonged activation of the inspiratory center in the medulla, but its action can be interrupted by afferent vagal impulses and to a lesser extent by the pneumotaxic centre.

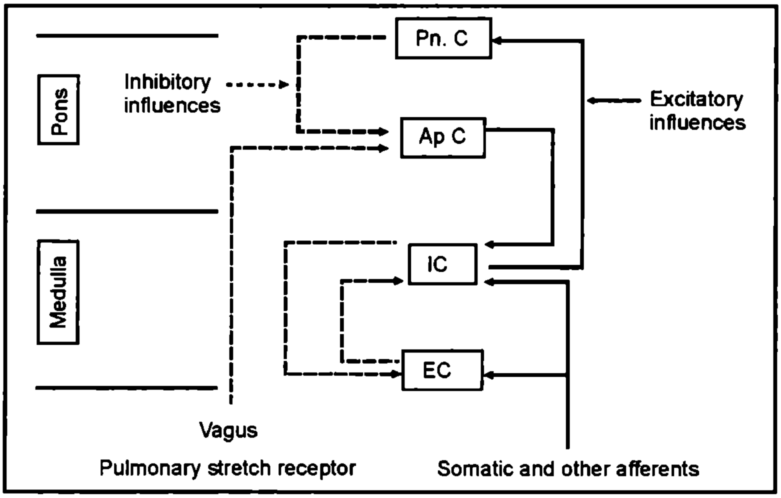


Figure 1.11: Possible organization of the respiratory center

3. *Pneumotaxic Centre*

   The pneumotaxic respiratory center, situated in the upper third of the pons, is capable of inhibiting the apneustic center. Stimulation of this centre results in tachypnoea. Some investigators believe that the role of this centre is the 'fine tuning' of the respiratory rhythm because normal rhythm can exist in the absence of this centre. The pneumotaxic centre has no inherent rhythm but seems to act by controlling the other centres.

Cortex

Breathing is under voluntary control to a considerable extent and the cortex can override the function of the brainstem within limits. It is not difficult to halve the arterial $PaCO_2$ by hyperventilation, although the consequent alkalosis may cause tetany with contraction of the muscles of the hand and foot (carpopedal spasm).

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There are three major components for the normal functioning of the respiratory system:

1. The neural and the muscular components (Fig. 1.12)
   The activity of the whole system depends on the initial excitation from both respiratory and non respiratory sources and also from the chemical stimuli such as the arterial carbon dioxide tension. Inspiration is initiated by the action of the apneustic centre and the somatic afferent impulses exciting the inspiratory center. Inspiratory activity causes nerve impulses to pass up the brain stem to the pneumotaxic center. These excite the pneumotaxic centre.

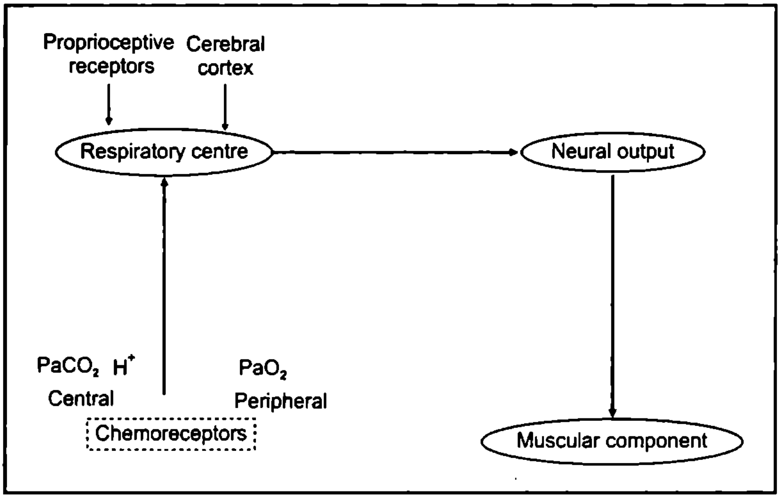


Fig. 1.12: Neural and the muscular components

   In other words, once inspiration is in progress the activity of the inspiratory centre is inhibited by the action of impulses from the pneumotaxic centre and from the pulmonary stretch receptors via the vagus nerve. This follows the inhibition of inspiration and allows expiration to take place.
   The neural output is influenced by input from the carotid (PaO_2) and central ($PaCO_2$, H^+) chemoreceptors, proprioceptive receptors in the muscles, tendons and joints, and impulses from the cerebral cortex. These impulses are governed by information from different receptors in the body.

2. The inherent properties of the lung, i.e. elastance and resistance. Normal gas exchange occurs if inspired gas is transmitted through structurally sound, unobstructed airways to patent, adequately perfused alveoli. Normally alveolar ventilation V_A and perfusion Q are well matched and proportional to the metabolic rate.

3. Diffusion across alveolar membrane
   The gas transfer takes place by a process of diffusion across the alveolar membrane.

CHAPTER 2

Respiratory Mechanics

Respiration is defined as the gas exchange between the organism and its surroundings. The primary role of the respiratory system is *to provide the body with oxygen ("fuel") and to remove carbon dioxide ("waste gas") from the alveoli which are in equilibrium with the blood (Fig. 2.1).*

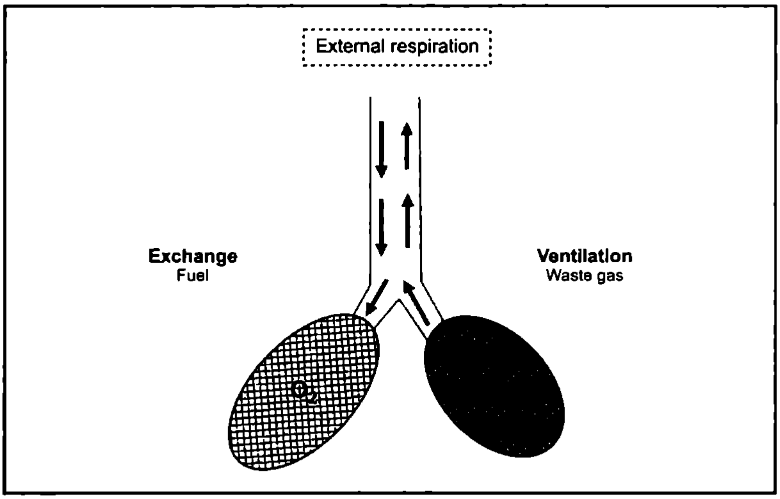


Figure 2.1: Primary role of the respiratory system

In other words the respiratory system maintains satisfactory partial pressure of oxygen and carbon dioxide in the arterial blood which exerts an influence on ventilation. The volume of ventilation is especially responsive to changes in carbon dioxide, though affected by both arterial PaO_2 and $PaCO_2$.

Peripheral chemoreceptors are in the carotid arteries and central chemoreceptors are close to the respiratory centre in the brain.

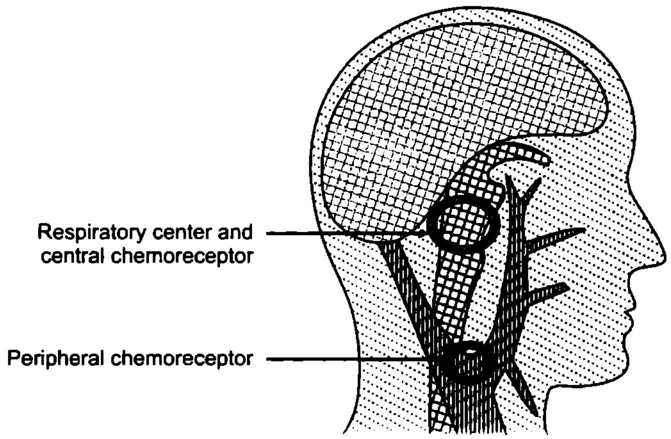


Figure 2.2: Type of chemoreceptors

Central chemoreceptors are bathed in brain extracellular fluid (ECF). CO_2 diffuses from the blood vessels to the cerebrospinal fluid (CSF), changes the pH values in the CSF surrounding the brain and the spinal cord, thus stimulating the chemoreceptor. However, H^+ and HCO_3 ions cannot easily cross the blood brain barrier.

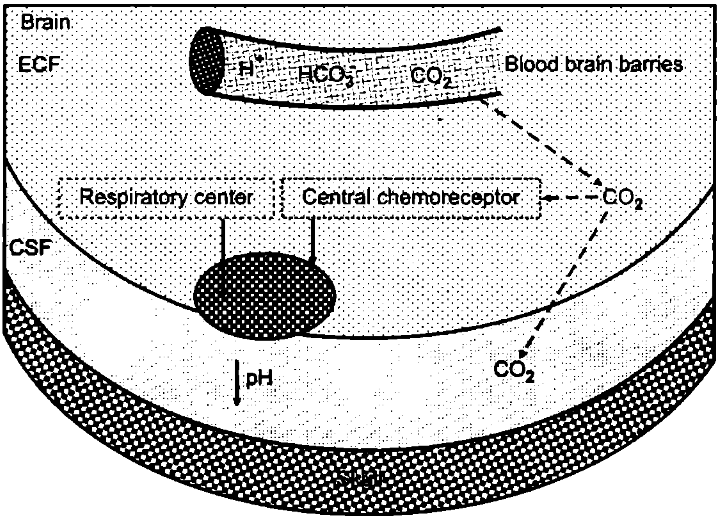


Figure 2.3: Environment of central chemoreceptors

NORMAL REGULATION OF BREATHING (FIG. 2.4)

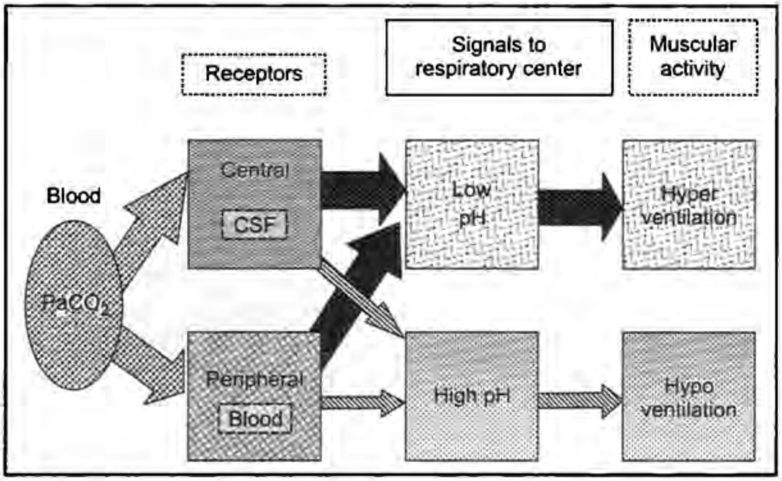


Figure 2.4: Normal regulation of breathing

Regulation at Breathing in Patient with Connecting Disease (Fig. 2.5)

The pH value of the CSF has a direct effect on the respiratory center in such a way that a low pH (high CO_2 level) stimulates breathing and a high pH (low CO_2 level) impedes breathing. In addition, the peripheral receptors are affected by the pH value of the blood in such a way that a low pH stimulates breathing. The volume and frequency of ventilation is determined by impulses from the respiratory centre in the medulla oblongata.

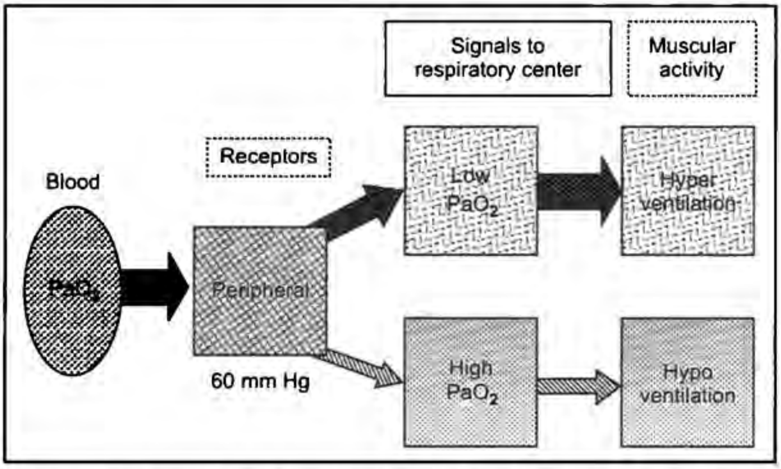


Figure 2.5: Regulation of breathing in a patient with chronic lung disease

In patients with chronic lung disease, due to chronic retention of CO_2 (low pH in CSF) over a long period of time, the respiratory center gets desensitized to the effects of raised $PaCO_2$. Hence, in these patients $PaCO_2$ levels have minimal or no influence in stimulating respiration. The impulses are then governed by the oxygen level in the blood, expressed as PaO_2 via the peripheral receptor, instead of $PaCO_2$. The respiratory center is stimulated only when PaO_2 is lowered to about 60 mm Hg.

Mechanics of Breathing

A balloon describes the procedure of inspiration and expiration.

A balloon may be filled in two ways:

1. *Positive pressure filling:* Air is pumped into the balloon. The air should be pumped at a pressure higher than that already existing in the balloon.
2. *Negative pressure filling:* Stretch the balloon to make it larger. In this method, air enters the balloon because the pressure in the balloon is lower during the process of stretching.

The mechanism responsible for the movement of air in the respiratory tract can be compared to the ways in which a balloon can be filled with air. Air moves into the lungs during inspiration and moves out of the lungs during expiration in response to pressure gradients created.

Inspiration

The air movement from the atmosphere to the lungs is accomplished by the work of respiratory muscles, when the patient is breathing spontaneously (Fig. 2.6).

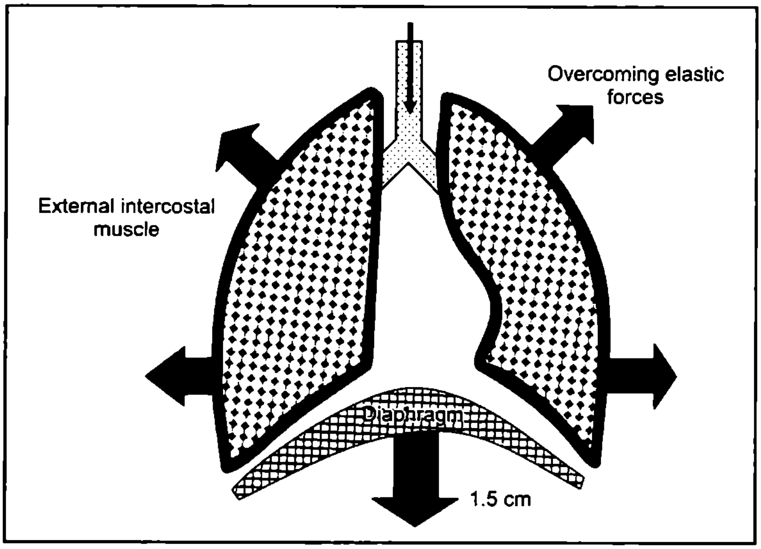


Figure 2.6: Inspiration

- Increase in size (volume) of the thoracic cage
  During inspiration, the primary ventilatory muscles cause the size (volume) of the thoracic cage to increase, overcoming the elastic forces of the lungs and the chest wall and the resistance of the airways. This is why inspiration is an active movement.
- Intrapleural pressure becomes more negative.
  As the volume of the thoracic cage increases, intrapleural pressure becomes more negative, resulting in lung expansion as the visceral pleura expands with the parietal pleura.
- Transairway pressure gradient.
  Gas flows from the atmosphere into the lungs as a result of the transrespiratory pressure gradient.

Expiration

During quiet breathing expiration is a passive process. Expiration is brought about by the relaxation of the inspiratory muscles (Fig. 2.7).

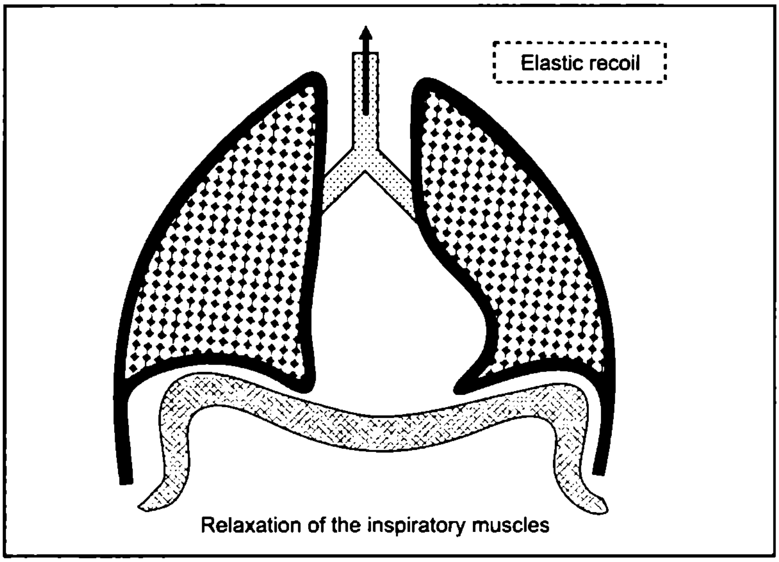


Figure 2.7: Expiration

The contraction of the inspiratory muscles increases the size of the thoracic cage; relaxation of the same muscles returns the thoracic cage to its original size. This is enough to generate sufficient positive pressure in the lungs to expel the normal tidal volume.

Air is driven out of the lungs by the elastic recoil of the lungs and chest wall as they return to their original position after inspiration. Exhalation occurs as a result of greater pressure at the alveolus when compared to atmospheric pressure (Fig. 2.8).

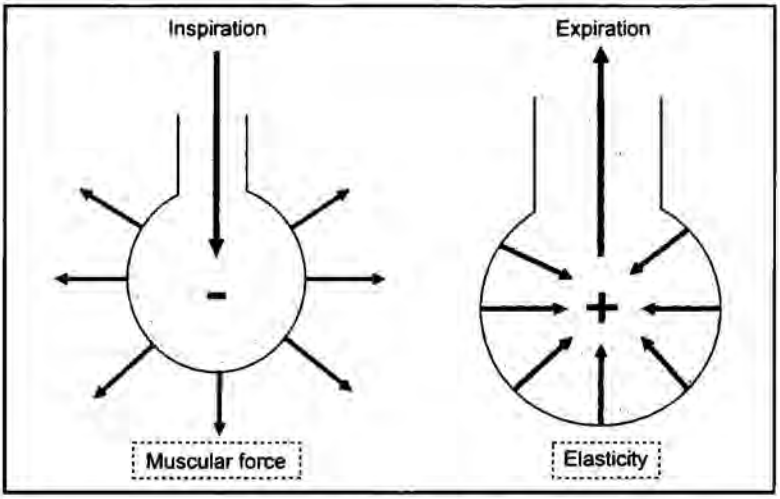


Figure 2.8: Inspiration and expiration

RESPIRATORY MUSCLES

Muscles of Inspiration

Diaphragm

The most important respiratory muscle is the diaphragm, a flat dome shaped muscle attached to the lower ribs. Inspiration is brought about by the downward movement of the diaphragm which increases the size of the thoracic cage from above downward. Since the downward movement of the diaphragm compresses the abdominal contents, the abdominal wall moves outwards during inspiration.

It moves about 1.5 cm during a normal breath, but can move up to 10 cm with deep breathing. In normal circumstances a movement of 1 cm downwards of the diaphragm causes about 350 ml of air to enter the lungs and thus normal tidal exchange of 500 ml per breath is accompanied by a movement of about 1.5 cm.

External Intercostal Muscles

Contraction of external intercostal muscles raises outwards outwards. The shape of the ribs is such that their elevation expands the chest, both transversely (bucket handle movement) and anteroposteriorly (pump handle movement). Thus, the combined action of the diaphragm and the external intercostal muscles increases the dimensions of the thoracic cage in all three directions. However, during normal, quiet breathing, the diaphragm alone is responsible for almost the entire inspiratory effort. On the other hand, during very forceful inspiration, besides the diaphragm and external intercostals, the sternocleidomastoid, scalene and serratus anterior muscles (accessory muscles of respiration) also contribute to the inspiratory effort.

Muscle of Expiration

- *Internal Intercostal Muscle:*
  The internal intercostal muscle helps to reduce the volume of the thoracic cage during active expiration by pulling the rib downwards.
- *Abdominal Muscles:*
  Forceful expiration or if expiration is obstructed. During forceful expiration or if expiration is obstructed, abdominal muscles play an important role in augmenting the expiratory pressure. These muscles include the abdominal recti, internal and external oblique and transverse abdominus muscles.

If expiration is obstructed, e.g. by an obstruction in the airway, the abdominal muscles and the muscles joining the inner surface of the ribs (internal intercostal muscles) help to draw the chest wall downwards and inwards and expel the air.

The volume of air that moves in and out of the lungs depends upon the characteristics of the lungs and airways and is independent of the force that is responsible for movement.

These include: respiratory pressure, compliance, resistance, elastance, energy required for ventilation (work of breathing).

RESPIRATORY PRESSURES

Intrapulmonary Pressure or Alveolar Pressure

This is pressure within the lungs. When the glottis is open, alveolar pressure is not very different from the pressure in the airways.

It is –1 cm H_2O at the peak of inspiration and a +1 cm H_2O at the peak of expiration, during quiet breathing. A healthy person can generate a pressure of 60 to 100 mm Hg (negative or positive) with maximal effort by the respiratory muscles.

Interpleural Pressure

It is absolutely essential to understand interpleural pressure which is the pressure between two pleural surfaces. The thoracic cage is lined on the inside with a serous membrane called the parietal pleura. The lungs too are covered with serous membrane called visceral or pulmonary pleura. Between the two layers of pleura is a very narrow space called the pleural cavity. The pleural cavity is filled with a thin layer of fluid which makes movement of the two pleural surfaces against each other smooth.

The normal pressure in the pleural space or interpleural pressure is negative, i.e. sub-atmospheric, both during inspiration and expiration.

- *Pleural Pressure during Quiet Breathing*

  During quiet breathing, pleural pressure fluctuates between -5 and -8 cmH_2O depending on the phase of respiration. However, pleural pressure becomes positive (i.e. above atmospheric) only during forceful expiration. Pleural pressure is always negative; more negative during inspiration and less negative during expiration, but always negative during quiet breathing.

- *Factors Causing Negative Intrapleural Pressure*

  The lungs would have a tendency to collapse while the thoracic cage would have a tendency to expand if they were not attached to each other. This does not happen in the body due to the anatomic relationship of these two structures. When the lungs are enclosed in the chest wall and attached to it by a two layered pleura, these tendencies create an inward pull on the visceral pleura and an outward pull on the parietal pleura since both, the thoracic cage and the lungs, are elastic structures (Figs 2.9 and 2.10).

Both the structures have a certain resting position at which there is no strain and attain a new position, because the lungs are expanded

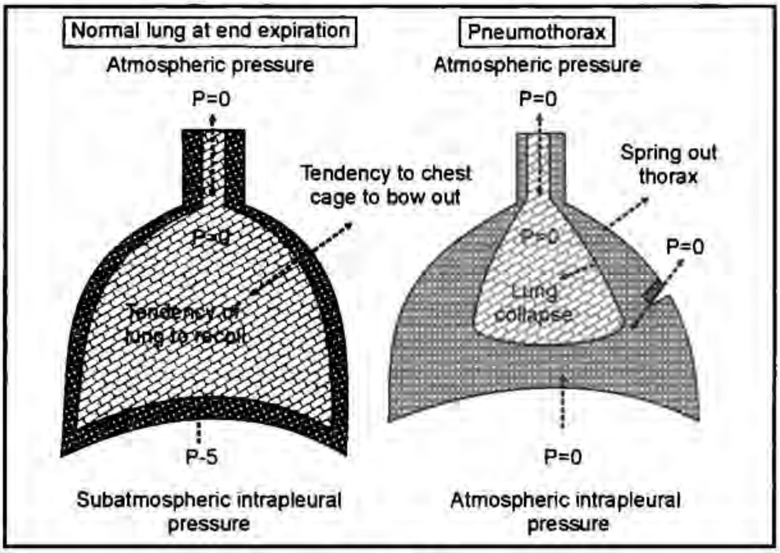
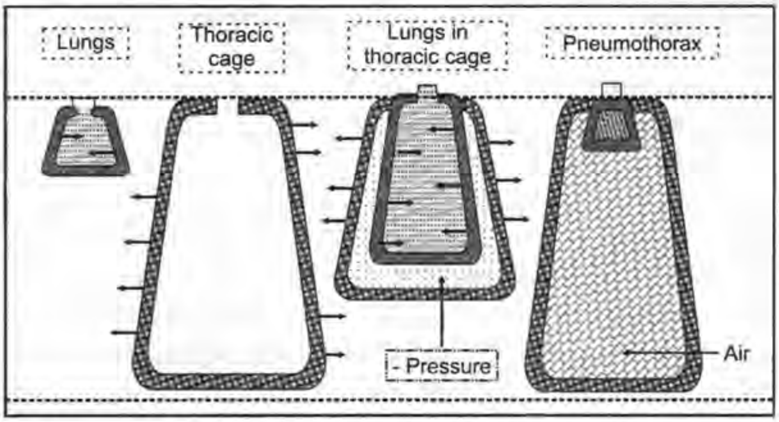

Normal lung at end expiration
Atmospheric pressure
P=0
P=0
Tendency of lung to recoil
Tendency to chest cage to bow out
P-5
Subatmospheric intrapleural pressure
Pneumothorax
Atmospheric pressure
P=0
Spring out thorax
P=0
Lung collapse
P=0
P=0
Atmospheric intrapleural pressure

Figure 2.9



Figures 2.9 and 2.10: The natural tendency of movement of the lungs and the thoracic cage, and their relationship in the body

by the elastic forces developed by the chest wall and the chest wall is pulled in by the elastic forces of the lungs. In this resting position there is a tendency of the lungs to recoil to its deflated volume and of the chest cage to retract outwards. The result of these opposing pulls manifests as a negative pressure in the space between the pleural surfaces (intrapleural space). This intrapleural negativity (subatmospheric pressure) helps to keep the lungs in an open position.

As always, a negative pressure can be sustained only in airtight structures. That is why, in any injury to the interpleural space giving rise to pneumothorax, the lung collapses because it is no longer pulled by the chest wall. The thoracic cage expands and air enters the interpleural space so that the interpleural pressure equalizes with the atmospheric pressure.

Transpulmonary/Transrespiratory Pressure

During spontaneous respiration, a pressure gradient is created between the proximal airway and the pleural space due to diaphragmatic and inspiratory muscle contraction, resulting in gas flow into the lungs. Another important pressure, i.e. transairway pressure is to be considered is the difference between the alveolar pressure and the atmospheric pressure.

Pulmonary Capillaries

Transmural Pressure

The pressure difference between the inside and outside of the capillaries is called transmural pressure. The pulmonary capillaries are unique, that is, they are surrounded by gas. There is a very thin layer of epithelial cells lining the alveoli but capillaries receive little support from this and consequently are liable to collapse or distend, depending on the pressure within and around them. The around pressure is alveolar pressure. When alveolar pressure rises above the pressure inside the capillaries, they collapse.

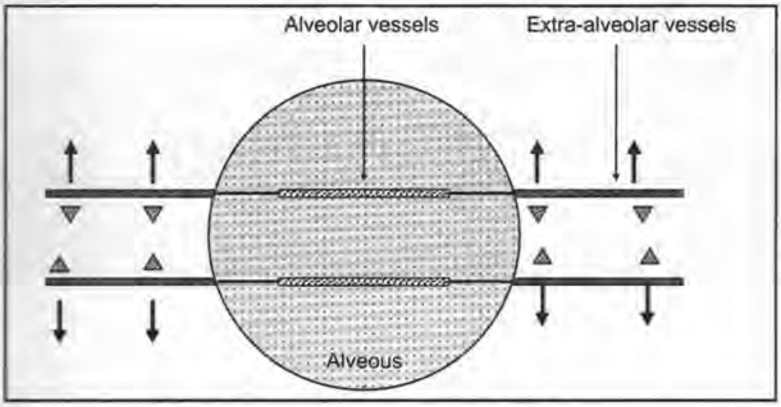


Figure 2.11: Alveolar and extra-alveolar vessels

- *Alveolar vessels (Fig. 2.11)*
  Alveolar vessels are exposed to alveolar pressure and include the capillaries and slightly larger vessels in the corner of the alveolar walls. Their calibre is determined by the relationship between the alveolar pressure and the pressure within them.
- *Extra-alveolar vessels (Fig. 2.11)*
  The extra-alveolar vessels are pulled by the radial traction of the surrounding lung parenchyma. And the effective pressure is lower than alveolar pressure. The pressure around the pulmonary arteries and veins can be considerably less than the alveolar pressure. As the lungs expand, these larger blood vessels are pulled open by the radial traction of the elastic lung parenchyma which surrounds them. Both, the arteries and veins, increase their calibre as the lung expands.

The behaviour of the capillaries and the larger blood vessels is so different that they are often referred to as alveolar and extra-alveolar vessels. Extra-alveolar vessels include all the arteries and veins that run through the lung parenchyma. Their calibre is greatly affected by lung volume because this determines the expanding pull of the parenchyma on their walls. The very large vessels near the hilum are outside the lung substance and are exposed to intrapleural pressure.

CHAPTER 3

Applied Respiratory Physiology

Respiration is the biological process of oxygen and carbon dioxide exchange across permeable membranes (Fig. 3.1).

- *Respiratory exchange ratio:* RR (External respiration).
  The relationship of the volumes of carbon dioxide and oxygen exchanged per minute by the lungs is referred to as the respiratory exchange ratio.
- *Respiratory quotient:* RQ (Internal respiration).
  The same relationship at the tissue level is referred to as the respiratory quotient RQ.

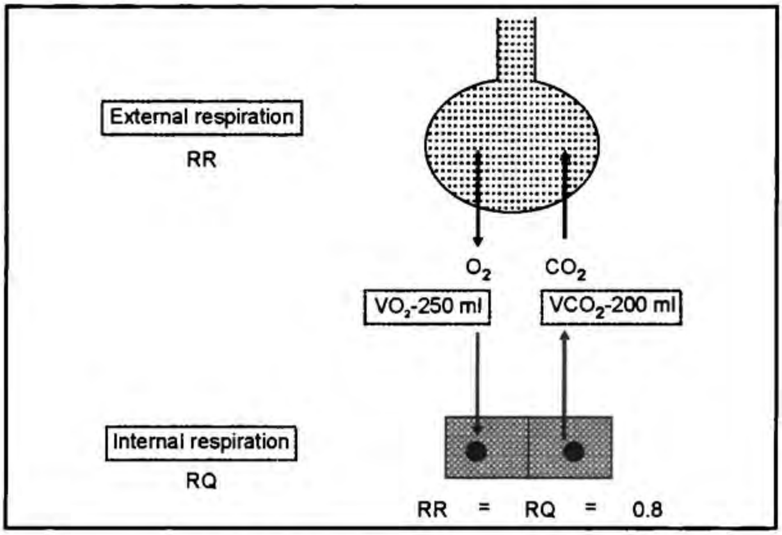


Figure 3.1: External and internal respiration

RESPIRATORY HOMEOSTASIS

Cardiovascular and pulmonary systems maintain internal stability through physiologic systems of coordinated feedback responses. Respiratory homeostasis requires that the RR and the RQ are equal. Gas values may sustain relatively unchanged because cardiovascular and pulmonary system increases work to maintain the homeostatic balance.

- The balance between the severity of disease and the degree of compensation by the cardiovascular and pulmonary system determines the degree of any abnormality in arterial gas values (Fig. 3.2).

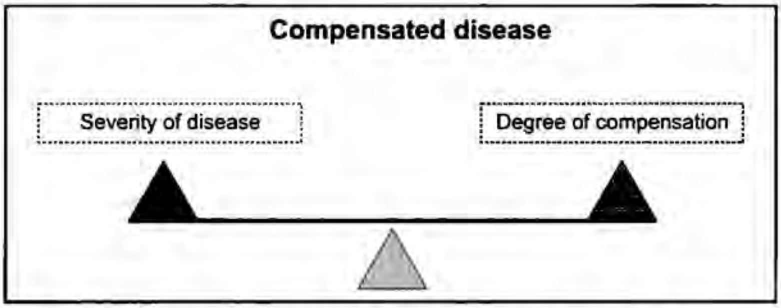


Figure 3.2: Normal arterial blood gases due to compensated disease

- Normal arterial blood gases do not mean there is an absence of disease because the homeostatic system can compensate.
- Abnormal arterial blood gas values reflect uncompensated diseases that may be life threatening (Fig. 3.3).

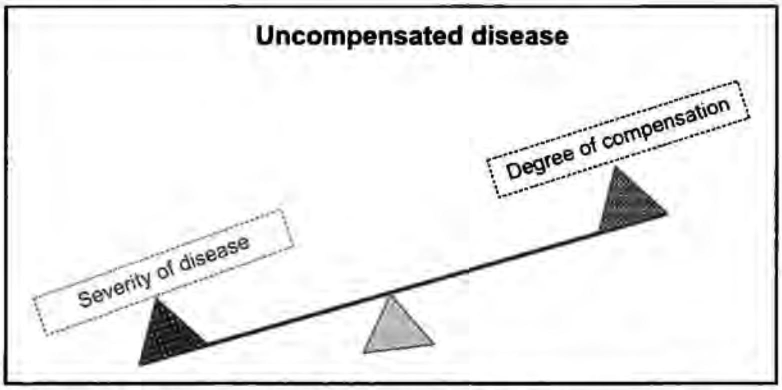


Figure 3.3: Abnormal arterial blood gases due to uncompensated disease

EXTERNAL RESPIRATION

External Respiration or Respiratory Exchange Ratio

This can be expressed in many ways:

- The respiratory exchange ratio represents the overall exchange capability of the lungs but not the individual variation within the different parts of the lungs.
- The relationship between the volumes of gas moving into an alveolus and the blood flow through the adjacent capillary is the best approach; however the disadvantage is the complexity of the concept. The matching of ventilation and perfusion is the method of expressing pulmonary gas exchange.

Regional Difference in Ventilation-perfusion Concept

A basic understanding of the alveolar ventilation-perfusion concept is essential to minimize the harmful effects of controlled ventilation and the clinical applications of blood gas measurements.

Regional Difference in Ventilation (Fig. 3.4)

Regional differences in ventilation refer to volume changes in relation to resting volume.

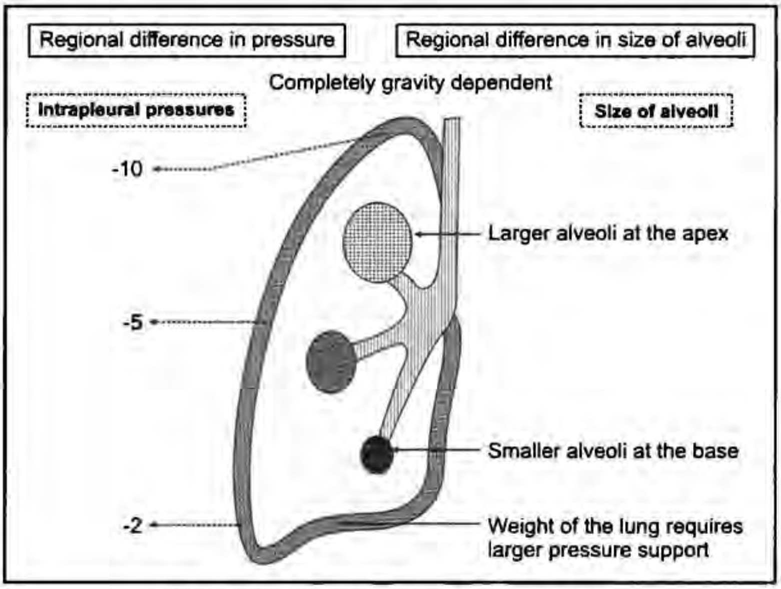

Regional difference in pressure
Regional difference in size of alveoli
Completely gravity dependent
Intrapleural pressures
Size of alveoli
-10
-5
-2
Larger alveoli at the apex
Smaller alveoli at the base
Weight of the lung requires larger pressure support

Figure 3.4: Regional difference in ventilation

a. *Regional difference in intrapleural pressure*

Intrapleural pressure is more subatmospheric at the apex than at the base which is completely a gravity dependent phenomenon.

- The most probable reason for this is that the lung is 'suspended' from the hilum and the weight of the lungs requires larger pressure support below the hilum than above.
- Thus, pressure near the base is greater and thereby the intrapleural negativity is less.
- Thus, at the base intrapleural negativity is less and pressure is greater than at the apex.

b. *Regional differences* in the size of alveoli in the upright position
In the upright position the lungs tend to sag towards the most dependent part under the influence of gravity so that the alveoli are smaller at the base than they are at the apex. This gravity dependence is true for any position.

c. *Regional difference in the transpulmonary pressure gradient during end of expiration (Fig. 3.5).*
Airway pressure and all alveolar pressure is zero (atmospheric) at the end of expiration. Intrapleural pressures are negative to a greater degree at the apex than at the base.

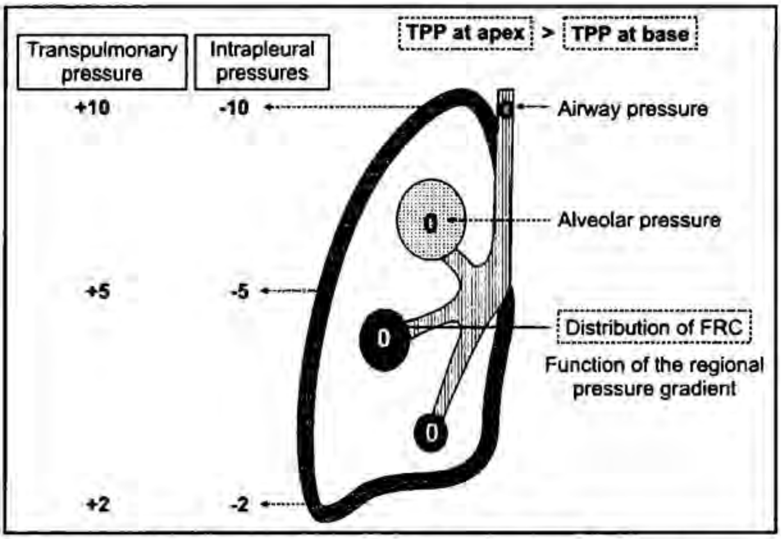


Figure 3.5: Regional difference in transpulmonary pressure gradient during end of expiration

- Approximately 40% of the total lung capacity (FRC) remains in the lung at the end of exhalation.
- The way this lung volume is distributed is primarily the function of the regional pressure gradients i.e. transpulmonary pressure (TPP) gradient across the lung.

- Transpulmonary pressure (airway pressure–intrapleural pressure) is greater at the apex than at the base. This is the primary reason why alveoli are larger at the apex and smaller at the base of the lungs.

d. *Regional difference in the transpulmonary pressure (TPP) gradient during inspiration*
  - When inspiration creates more subatmospheric pressure in the pleural space, the change in TPP at the base will be far greater than the change in TPP at the apex (Fig. 3.6).

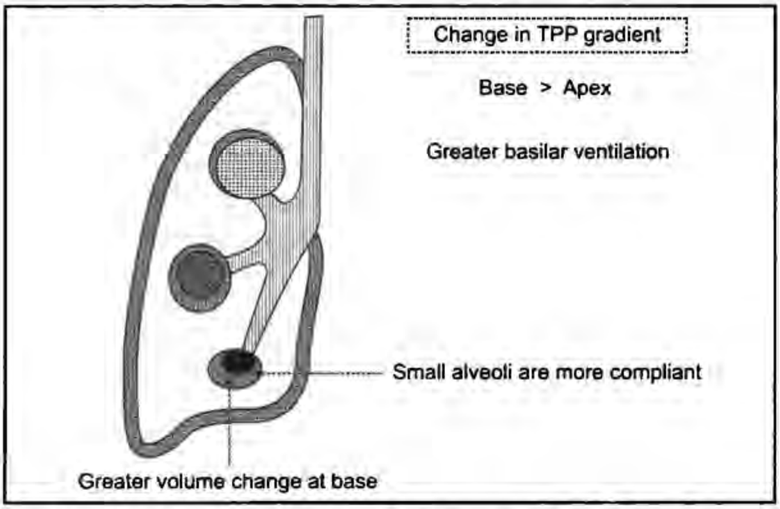

Change in TPP gradient
Base > Apex
Greater basilar ventilation
Small alveoli are more compliant
Greater volume change at base

Figure 3.6: Regional difference in TPP gradient during inspiration

  - Small alveoli are more compliant than larger ones so that during inspiration a greater volume change occurs in the former.
  - Thus, relatively more of the inspired tidal volume will tend to distribute to the basillar alveoli.

e. *Regional differences in ventilation for dependent lungs in any body position*
  - The change in ventilation decreases approximately linearly with the distance up the lungs being about twice as great at the base than at the apex.
  - Compared with the apex, the base of the lungs has a smaller resting volume and undergoes a larger volume change per unit of lung, therefore, basilar ventilation is greater.
  - This is true for dependent lungs in any body position. Ventilation is preferentially distributed to the dependent parts of the lung provided the basal areas are not so compressed that airway closure has occurred.
  - Also, the differences in regional ventilation become less during exercise and are reversed in the inverted position.

In Diseased Lungs

In diseased lungs the two main factors controlling the distribution of ventilation are:

- *Patency of airways*

  Any factor, which reduces the airway caliber and therefore raises the resistance to airflow, will result in a fall in ventilation to the affected region.
- *Local changes in compliance within the lungs*

  Similarly, any area in which the local compliance has decreased will be less ventilated than the surrounding more expandable normal lung tissue.

Regional Differences in Perfusion

The normal distribution of blood flow throughout the pulmonary vasculature is dependent primarily on gravity and cardiac output.

a. *Gravitational effects on blood flow (Fig. 3.7).*

   In a healthy person standing erect, there is a distance of approximately 30 cm from the apex to the base of the lungs. Assuming that the pulmonary artery enters the lungs halfway between the top and the bottom, the pulmonary artery pressure must be great enough to overcome a gravitational force of 15 cm H_2O to supply flow to the apex. A similar gravitational force would be adding flow to the base. It is seen that a column of blood (essentially water) of 15 cm height exerts a pressure of approximately 11 mm Hg.

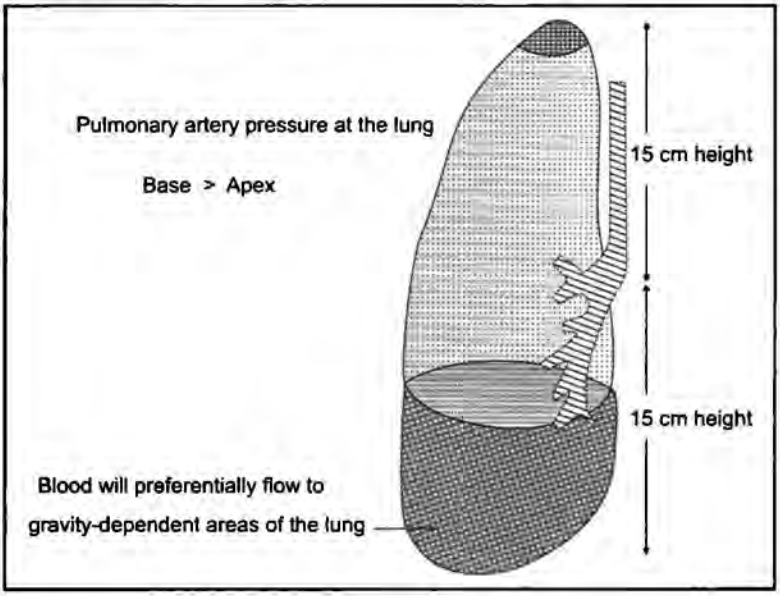

Pulmonary artery pressure at the lung
Base > Apex
15 cm height
15 cm height
Blood will preferentially flow to
gravity-dependent areas of the lung

Figure 3.7: Gravitational effects on blood flow

This gravitational effects on blood flow results in a pulmonary artery pressure at the lung base of greater magnitude than the pulmonary artery pressure at the apex. That is why blood will preferentially flow through the gravity-dependent areas of the lungs. This is true for any body position.

b. *Effects of gravity on pulmonary perfusion (Fig. 3.8)*
Under normal circumstances alveolar pressures are equal throughout the lungs while the vascular pressures vary throughout the lungs.

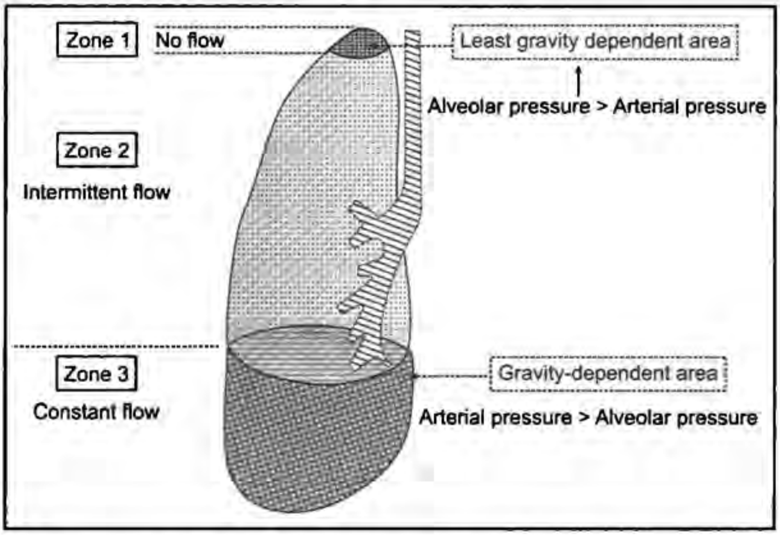

Zone 1
No flow
Least gravity dependent area
Alveolar pressure > Arterial pressure
Zone 2
Intermittent flow
Zone 3
Constant flow
Gravity-dependent area
Arterial pressure > Alveolar pressure

Figure 3.8: The three-zone model illustrating the effects of gravity on pulmonary perfusion

It has become well accepted to refer to the gravity effects on pulmonary perfusion in terms of the three-zone model.

Zone 1—Least gravity dependent area

- Zone 1 is the least gravity dependent area of the lungs and has alveolar pressures higher than the pulmonary arterial pressures, resulting in the virtual absence of blood flow. It should be noted that the total absence of pulmonary blood flow to these areas does not exist to any significant extent in the normally perfused lung.
- *Zone 2—Cardiac cycle is more important than the ventilatory cycle*
Zone 2 is an area of complex and varying intermittent blood flow. The extent of blood flow in zone 2 depends primarily on the relationship of pulmonary artery pressure to alveolar pressure. Under normal circumstances this is determined far more by the cardiac cycle than by the ventilatory cycle.

- *Zone 3—Gravity dependent area*
  Zone 3 is the gravity dependent area of constant blood flow (arterial pressure is always greater than alveolar pressure).
- *Significant absence of perfusion*
  However, if pulmonary artery pressure significantly decreases (hypoperfusion) or alveolar pressure significantly increases (positive pressure therapy), the absence of perfusion to the least gravity-dependent areas of the lungs can become significant.

  Cardiac output is the amount of blood ejected by the right ventricle per minute and is a major determinant of blood flow through pulmonary vasculature. In other words, the greater the cardiac output the greater the pulmonary artery pressures. Thus, in normal lungs, as the cardiac output decreases zone 2 and zone 3 decreases.

  Compared with the systemic vasculature the autoregulatory capability of the pulmonary vasculature is inferior. Cardiac output changes indirectly influence the distribution of the blood flow, significantly in the pulmonary vasculature than in the systemic vasculature by gravitational effects.

INTERNAL RESPIRATION

The O_2 and CO_2 exchange between systemic capillary blood and cells is referred to as internal respiration.

- The primary purpose of respiration is to provide oxygen to the cells.
- The secondary purpose is to remove carbon dioxide from the cells.

Respiratory Quotient

The RQ is the relationship of CO_2 production in 1 minute (VCO_2) to oxygen consumption in 1 minute (VO_2). The volume of CO_2 normally produced in 1 minute is approximately 200 ml and the volume of O_2 normally consumed in 1 minute is approximately 250 ml, therefore, the normal RQ is 0.8.

Basis of Internal Respiration

Internal respiration primarily depends on:

Regional Perfusion

The pulmonary system is essential to external respiration; the cardiovascular system is essential to both external and internal respiration. Perfusion which is inadequate to serve the O_2 demands will reduce internal respiration even when there is adequate pulmonary function.

Metabolism

The rate of metabolism normally varies greatly among different organ systems. Metabolic rates of different organ systems vary in the presence of disease and stress.

CHAPTER

4

Distribution of Ventilation

The total surface area of the alveolar epithelium is about 72-80 m^2. Approximately 85-95% (about 70 m^2) of this area is in contact with pulmonary capillaries, constituting the alveolocapillary interface. The inspired gas which is distributed at alveolocapillary interface will take part in ventilation or is wasted, and accordingly classified as:

- Effective ventilation or Alveolar ventilation
- Wasted ventilation or Dead space ventilation.

ALVEOLAR VENTILATION

Definition

Alveolar ventilation, or effective ventilation, is defined as that portion of tidal or minute volume, which takes part in gas exchange.

Alveolar Volume V_A

Alveolar volume V_A is the difference between tidal volume V_T and dead space volume V_D.

$$V_A = V_T - V_D$$

The equation shows that alveolar volume can be increased by raising the tidal volume or by reducing the dead space volume.

Minute Alveolar Ventilation

When the measurements are made over one minute it is called minute alveolar ventilation. It depends on the respiratory rate. Hypoventilation can result when the respiratory rate is too slow or absent.

Minute alveolar ventilation – $\dot{V}_A = (V_T - V_D) \times RR$

Normal value is about 3.5 to 4.5 lit/min in adults.

Alveolar Air Equation

$$\text{Alveolar oxygen tension} = \text{Inspired oxygen tension} - \frac{\text{Alveolar carbon dioxide tension}}{\text{Respiratory quotient}}$$

$$PAO_2 = PIO_2 - \frac{PACO_2}{0.8}$$

If you substitute normal figures in the equation, the equation is:

$$100 \text{ mmHg} = 150 - \frac{40}{0.8}$$

Clinical Importance of Administration of Minimum 33% Oxygen

Rise in arterial carbon dioxide tension will increase alveolar carbon dioxide tension. Thus, if the alveolar carbon dioxide tension rises the alveolar oxygen tension will fall. This will result in a fall in arterial oxygen tension. In other words, a rise in $PaCO_2$ will result in arterial hypoxaemia with spontaneous ventilation. This arterial hypoxaemia will be exaggerated due to other changes in the lungs under anesthesia.

That is why it is always advisable to administer at least 33% (atmospheric oxygen is 21%) oxygen in all anaesthetic gas mixtures. This compensates for any rise in $PaCO_2$, and also for the other pulmonary changes which occur under anesthesia.

For example, if 30 percent oxygen is administered, the equation becomes:

$$155 \text{ mm Hg} = 230 - \frac{60}{0.8}$$

There are always dangers of hypoventilation during anesthesia. It is not sufficient to oxygenate a patient by raising the inspired oxygen percentage, but adequate steps must be taken at the same time to remove the carbon dioxide. It is important to remember that just because a patient is having a good color does not mean he is having normal alveolar ventilation.

Hypoventilation means a reduction in the bulk flow of air into the lungs. Clinically, hypoventilation is characterized by a reduction of alveolar ventilation producing impaired ventilation of an alveolus which leads to impaired oxygenation and increase in arterial carbon dioxide tension giving rise to type II respiratory failure. The resulting rise in alveolar carbon dioxide tension will produce a fall in alveolar ventilation unless extra oxygen is added to the inspired air.

Clinical Assessment of Alveolar Ventilation

- Observation of the reservoir bag
- Movements of the chest and abdomen
- Respiratory rate
- Color of blood in the patient's capillary bed
- Measurement of minute volume using respirometer

Steps to Improve Minute Alveolar Ventilation in Ventilated Patient

By Increasing the Alveolar Volume

In mechanical ventilation, a decrease in alveolar volume occurs when the tidal volume to the patient is decreased or the dead space volume is increased.

- Hypoventilation caused by a reduction in tidal volume can be corrected by increasing the tidal volume on the ventilator.
- Though the tidal volume can be corrected, dead space volume is difficult to change because anatomic dead space is rather constant.
- Physiological dead space which is caused by a lack of perfusion, i.e. alveolar dead space requires a different strategy which should improve blood flow.

Minute ventilation = Tidal volume x respiratory rate

Minute ventilation is inversely proportional to the $PaCO_2$. Minute ventilation can be altered by increasing /decreasing either tidal volume or frequency depending on blood gases. Minute ventilation also affects the PaO_2 nonlinearly. Manipulating the minute volume for the purpose of achieving a change in PaO_2 is undesirable, because only small changes in PaO_2 can be brought about by large alterations in minute ventilation. Such large changes in minute ventilation have a profound and unwanted effect on PaO_2. The effects produced on $PaCO_2$ as a result of altering tidal volume can be different from those achieved by altering respiratory rate.

Example - I

Minute ventilation 5000 ml/min = 500 × 10

Alveolar ventilation 3500 ml/min = (500-150) × 10

Example - II

Minute ventilation 5000 ml/min = 250 × 20

Alveolar ventilation 2000 ml/min = (250-150) × 20

The alveolar ventilation would exceed in example I.

It is alveolar ventilation that determines the $PaCO_2$, however if minute ventilation is to be increased, it should be increased carefully. $PaCO_2$ is inversely proportional not to all parts of minute ventilation, but to the part of ventilation which takes part in gaseous exchange, i.e. Alveolar ventilation.

By Increasing the Respiratory Rate

Hypoventilation due to a reduction in the respiratory rate can be corrected by increasing the respiratory rate on the ventilator.

$PaCO_2$ measurement is essential to **monitor ventilatory status**

$$\dot{V}_A = \frac{VCO_2}{PaCO_2}$$

VCO_2 - Carbon dioxide produced by cellular metabolism in one minute.

The equation shows the inverse relationship between alveolar ventilation and $PaCO_2$ assuming that VCO_2 is constant. Thus, when minute alveolar ventilation is low, typical finding in blood gas analysis is an elevated $PaCO_2$. That is why a patient's ventilatory status can be best monitored by $PaCO_2$ measurement. Pulmonary factors control the excretion of carbon dioxide by the lungs and it is directly related to the V_T, physiological dead space and the respiratory rate.

$$\dot{V}_A = (V_T - V_D) \times RR$$

Causes of Insufficient Alveolar Ventilation

1. Insufficient breathing (low minute ventilation)
   - *Depression of the respiratory centre*
     Deep anesthesia, respiratory depressant drugs such as overdose of sedatives, infection, cerebral hypoxia and muscle relaxants, all tend to depress alveolar ventilation.
   - *Paralysis of respiratory muscles*
     Muscular disease, muscle relaxants, trauma can paralyze respiratory muscles.
   - *Improperly set ventilator*
     - Inadequate ventilation according to the patient's current condition.
     - Inadequate time for gas exchange (inspiratory to expiratory time ratio) Small tidal volumes with increased dead space ventilation.
2. Increased dead space

DEAD SPACE VENTILATION

The volume of the inspired gas that does not come into contact with the pulmonary capillary is termed dead space volume. Dead space ventilation is defined as wasted ventilation or a condition in which ventilation is in excess of perfusion. It is expressed as the product of dead space volume and the respiratory rate. Dead space ventilation therefore, increases linearly with breathing frequency.

Dead space ventilation = $V_D \times f$

Alveolar ventilation = $(V_T - V_D) \times f$

COMPENSATION FOR INCREASED DEAD SPACE VENTILATION

- *Increasing tidal volume:*
  Increasing the tidal volume can compensate for an increased dead space ventilation.
- *Increasing the respiratory rate along with increase in tidal volume:*
  Increased dead space ventilation can also be compensated by increasing the respiratory rate provided the tidal volume is also increased simultaneously. But if the respiratory rate increases and the tidal volume decreases, dead space ventilation increases at the expense of alveolar ventilation. If the tidal volume is not increased, the increased respiratory rate will lead to an increase in the work of breathing.

Dead Space

Dead space is that portion of the respiratory system in which no significant gas exchange occurs between blood and air.

Types of dead space are as follows:

1. Physiological dead space (V_D phys) or Functional dead space
   - Anatomical dead space
   - Alveolar dead space
2. Apparatus dead space

Physiological Dead Space (V_D phys)

It is defined as that fraction of the tidal volume which is not available for gaseous exchange. The physiological dead space is about 2 ml/kg body weight. In an intubated patient it is about 1.25 ml/kg. Under normal conditions, physiological and anatomical dead spaces are almost equal and amount to one third of the tidal volume. In diseased conditions where alveolar dead space ventilation is increased, physiological dead space is always higher than anatomical dead space. It includes therefore, not only the anatomical dead space, but those alveoli which are ventilated but not being perfused as in these alveoli gaseous exchange does not occur. If areas of the lungs are over-ventilated and normally perfused, they too contribute to an increase in dead space.

Clinical conditions that increase physiological dead space are as follows:

- *Decreased tidal volume*
  - Drug overdose, neuromuscular disease
- *Increased alveolar dead space*
  - Decreased cardiac output, e.g. congestive heart failure, blood loss, hypovolaemia, hypotension.
  - Obstruction of the pulmonary blood vessels, e.g. pulmonary vasoconstriction, pulmonary embolism, etc.

Anatomical dead space (V_D anat) or airway dead space: Anatomical dead space is a gas transport zone and not a gas exchange zone (Figs 4.1 and 4.2).

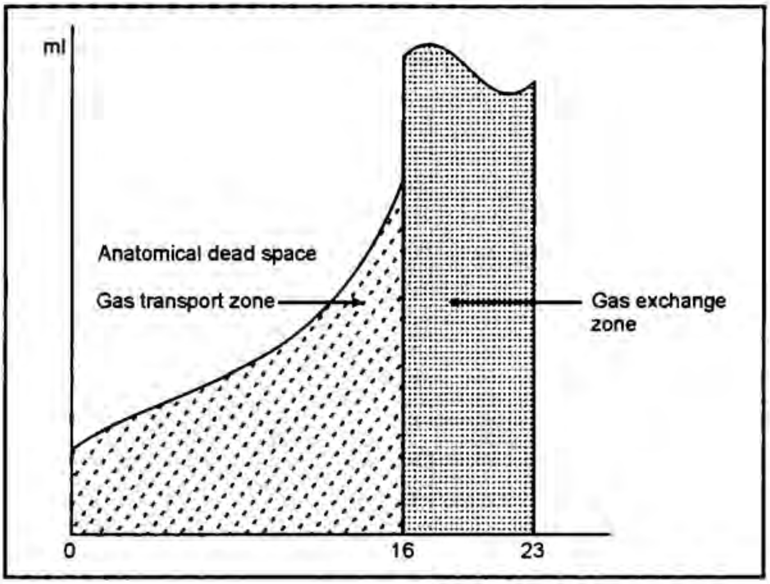
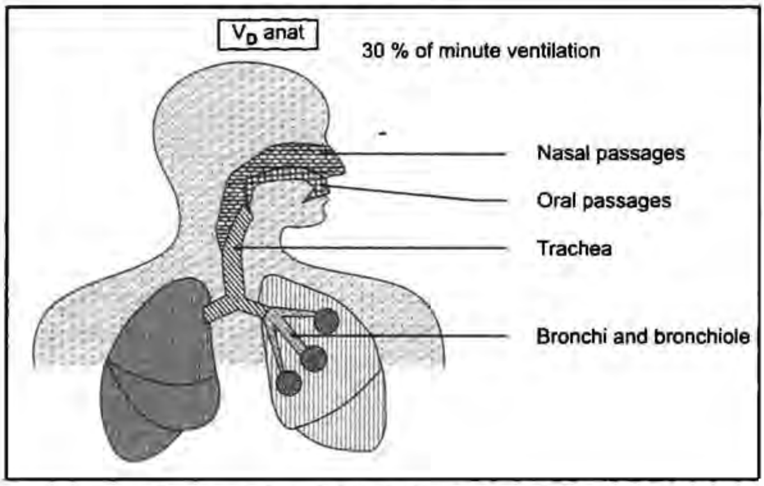


Figure 4.1



Figures 4.1 and 4.2: Anatomical dead space

It comprises the volume of the respiratory passages or conducting airways, extending from the nostrils and the mouth down to the terminal bronchi. Conducting airways conduct air down to the respiratory zone of the lung, but play no role in gas exchange. It includes the nasopharynx, the trachea, the bronchi and the bronchioles but not the alveoli. Its air-filling volume is referred to as dead space volume.

Factors Influencing Anatomical Dead Space Volume

- Age and sex
  Its capacity varies with age and sex. Normally it is 150 ml, but in young women it may be as low as 100 ml, while in old men it can rise to as much 200 ml (Old age - CC > FRC)
- The height and weight of the individual/Position
- Flexion and extension of the neck (Fig. 4.3)

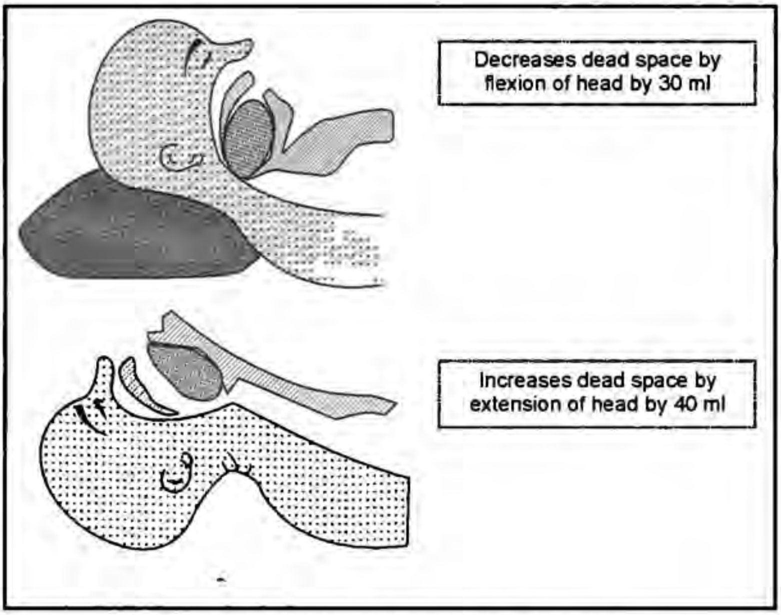

Decreases dead space by flexion of head by 30 ml
Increases dead space by extension of head by 40 ml

Figure 4.3: Flexion and extension of the neck

Depression of the lower jaw with flexion of the head (a common cause of respiratory obstruction in the anesthetized patient) reduces the dead space by 30 ml. On the other hand, protrusion of the jaw with extension of the head increases dead space by 40-ml.

- Pneumonectomy also reduces dead space.
- Intubation reduces anatomical dead space by 70 ml and tracheostomy by 90 ml or 60%.

Normally, the conducting airways contribute to about 30% of tidal volume. For a tidal volume of 500 ml, about 150 ml of the volume is wasted since it does not take part in gas exchange. This anatomical dead space is estimated to be about 1 ml per pound of ideal body weight in non-intubated patients.

Alveolar dead space (Fig. 4.4)

Alveolar dead space is the volume of gas reaching non-perfused alveoli and areas of alveoli which are over-ventilated and normally perfused. The alveolar dead space is minimal in spontaneously breathing, healthy individuals.

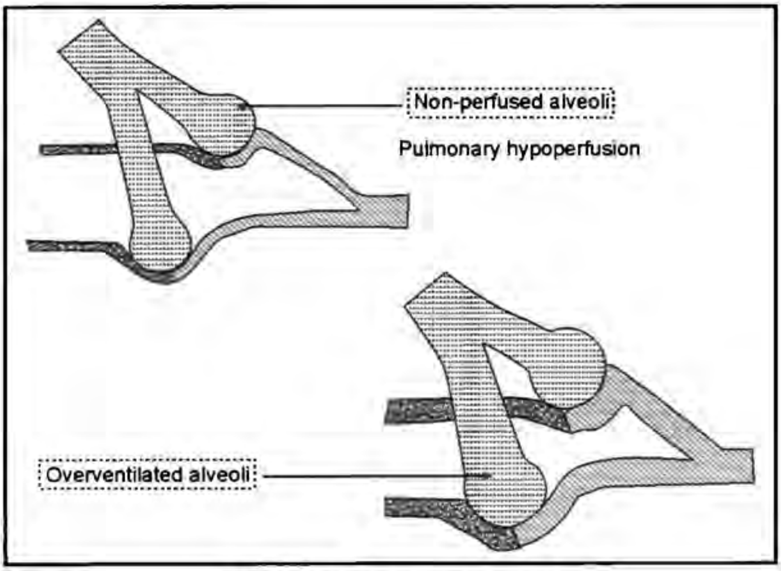


Figure 4.4: Alveolar dead space

Factors increasing alveolar dead space are as follows:

1. *IPPV*

   During positive pressure ventilation, alveolar dead space increases markedly (even in the healthy individual) and can amount to ¼ of the alveolar ventilation. This is because of the undesirable effect of increased intrathoracic pressure producing ventilation/perfusion imbalance. Anesthetic agents and pre-existing lung diseases can further increase this effect.

2. *Lateral posture-mechanical ventilation*

   A ventilation perfusion mismatch can be observed in patients in a lateral posture producing increase in alveolar dead space:

   - The non-dependent lung will have well-ventilated compartments with insufficient perfusion.

- The dependent lung will have poorly ventilated but well perfused compartments during mechanical ventilation.

3. *Decreased pulmonary perfusion* (Fig. 4.5)

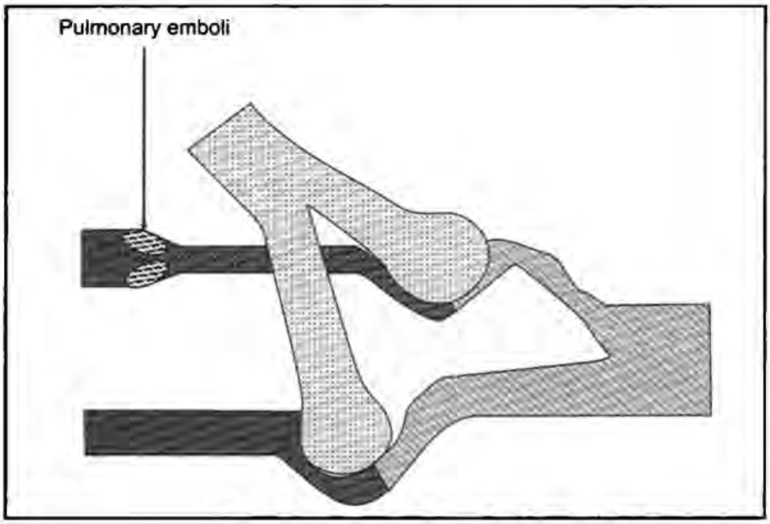


Figure 4.5: Pulmonary embolus

It is the normal lung volume that is unable to take part in gas exchange because of reduction or lack of pulmonary perfusion, e.g. pulmonary embolism, hamorrhage. This ventilation is therefore wasted, appearing as an increase in physiological dead space. A large alveolar dead space is also seen in emphysema.

4. *Improper ventilatory settings*
   - Large tidal volume
   - High respiratory rates
   - When inspiratory time is reduced to 0.5 seconds or less during controlled ventilation

Dead Space Quotient—V_D/V_T

Normally dead space ventilation involves 30% and alveolar ventilation involves about 70 % of the tidal volume, i.e. the dead space quotient = 0.3. In normal individuals, dead space is mostly anatomical dead space and dead space ventilation is inversely proportional to the tidal volume. In normal individuals V_D remains constant, and increase in V_T will cause a fall in the ratio of V_D to V_T.

Decrease in the tidal volume causes a higher anatomical dead space to tidal volume percent. Using the above example, the dead space to tidal volume percent would increase from 30% (150/500) to 50%

(150/300) if the tidal volume was decreased from 500 ml to 300 ml.

150/500 = 0.3 or 30%

150/300 = 0.5 or 50%

$V_D/V_T = 33 + \text{Age}/3$ percent

V_D/V_T ratio: normal 0.25 to 0.4

In ventilated individuals dead space quotient can be high due to increase in the anatomical dead space (ventilatory circuit – circuit compressibility, heat moisture exchangers) or increase in alveolar dead space (decrease in cardiac output) or both. Postoperatively, especially after laparotomies and thoracotomies, the dead space quotient is approximately 0.5 and can increase to 0.55 within the first 24 hours.

$$V_D = V_T \times \left(\frac{PaCO_2 - P_ECO_2}{PaCO_2}\right)$$

A measurable increase in the dead space quotient means that in some lung areas the perfusion almost ceases although the area is still being ventilated.

Apparatus Dead Space (Fig. 4.6)

Some of the inspiratory gas does not participate in gas exchange. This gas is mixed with expiratory gas in connection tubes, hygroscopic humidifier and other accessories. Apparatus dead space is defined as

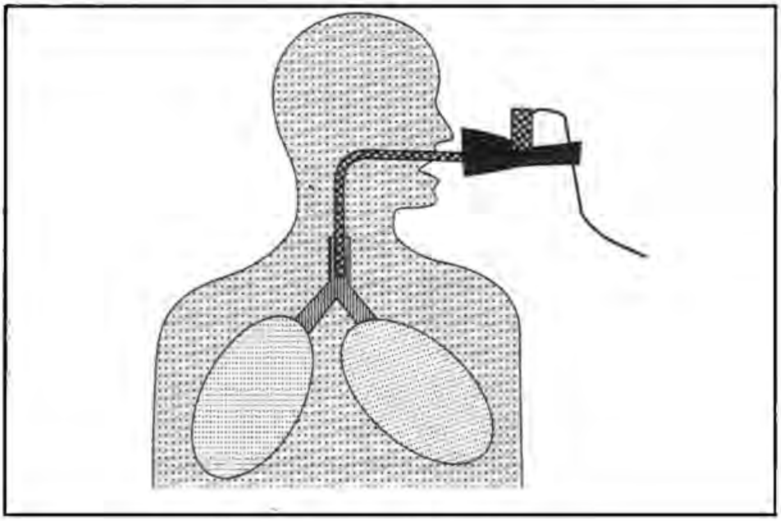


Figure 4.6: Apparatus dead space

the volume of gas contained in these passages. It consists of the volume of gas contained in any anaesthetic apparatus between the patient and that point in the system where re-breathing of exhaled carbon dioxide ceases to occur (e.g. the expiratory valve in a Magill system or the side arm in an Ayer's T-piece).

The volume of an adult facemask with connections up to the point of the expiratory valve is 125 ml. The apparatus dead space should always be added to the required tidal volume, in order to achieve adequate alveolar ventilation.

Importance of the Pattern of Gas Flow and Channelling of the Gases

The pattern of the gas flow and channelling of the gases within the apparatus reduces the role of dead space. In practice, physiological mechanisms react to these changes and tend to return $PaCO_2$ to normal. The central core of the gas stream moves rapidly whilst gas in the periphery moves relatively slowly. Thus, the central element represents the major portion of the ventilation, thus reducing the role of dead space.

VENTILATORY FAILURE

Ventilatory failure is the inability of the pulmonary system to maintain the proper removal of carbon dioxide. Hypercapnia is the feature of ventilatory failure. When carbon dioxide production exceeds its removal it results in respiratory acidosis.

Hypoxaemia can be the secondary complication of ventilatory failure but this type of hypoxaemia usually responds well to oxygen therapy. Without supplemental oxygen the degree of hypoxaemia corresponds to the severity of ventilatory failure.

CHAPTER 5

Compliance

DEFINITION

Compliance is the ability of the elastic structure to change its volume to applied pressure; therefore it is a measure of elasticity and expansibility. The lungs and the thoracic cage are both elastic structures which display a constant relationship between distending pressure and change in volume. The volume of air that moves into the lungs is not only influenced by the compliance and but also by the alveolar pressure range.

The change in volume per unit change in pressure is called compliance.

$C = \Delta V/\Delta P$

Compliance has two components

1. *Lung compliance (C_L or C_{pulm}) Figure 5.1:*
   Lung compliance is a volume change (ΔV) in the lung per unit pressure change (ΔP). Pressure change is the difference in alveolar pressure and the pleural pressure.
   It is called transalveolar pressure and determined primarily by the elastic recoil of the lung tissue. Lung compliance is assumed to be equal to the chest wall compliance.
   C_L or Cpulm - 200 ml/cm H_2O
2. *Chest wall compliance (C_{CW}) Figure 5.2:*
   If we imagine a chest wall containing no lungs within, the volume change that occurs within the chest for a unit change in pressure is the chest wall compliance.

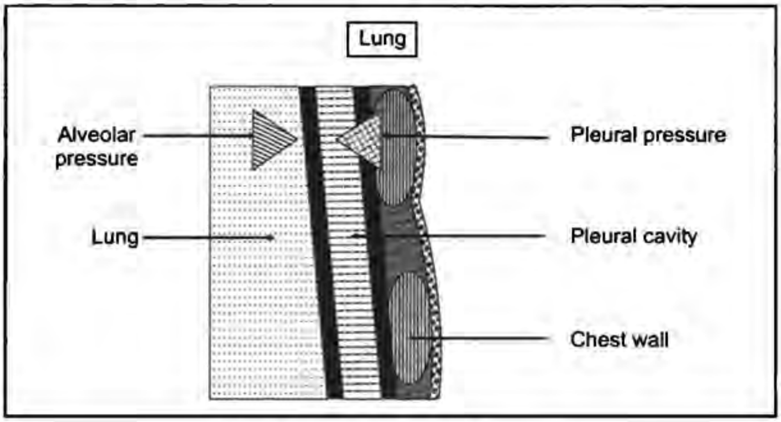


Figure 5.1: Transalveolar pressure

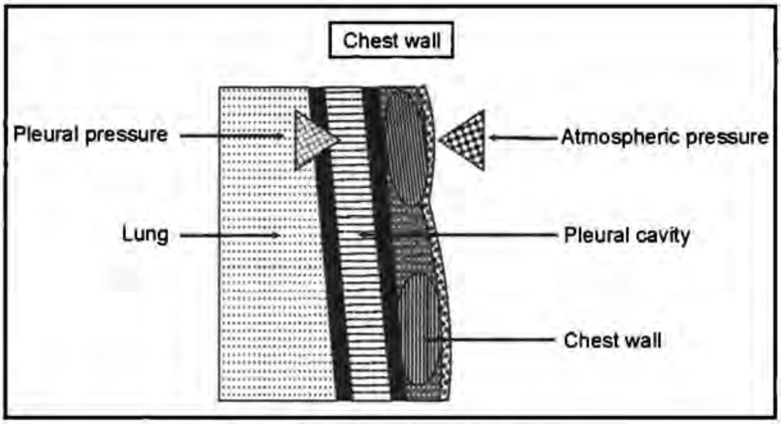


Figure 5.2: Transpleural pressure

The pressure change is the pressure difference between the pleural cavity and the outside (atmospheric) pressure which is called transpleural pressure. The transpleural pressure is a function of the elastic recoil of the chest cage.

Normal chest wall compliance – 200 ml/cm H_2O

Total Compliance (C_{TOT}) (Fig. 5.3)

Compliance of the total respiratory system is less than that of the lungs or the thoracic cage individually, because they normally work at cross purposes. The lungs try to pull the thoracic cage inwards while the thoracic cage tries to pull the lungs outwards. That is why it is more difficult to distend the lungs and thoracic cage together than either of them individually.

Normally a pressure change of 1 cm of water achieves a volume change of 200 ml in the lung if there is no chest wall. But when the same pressure is used to overcome the elastic forces of the chest wall in addition to the elastic forces of the lungs, it can achieve only half the volume change. This pressure change is the pressure difference between the alveoli and the atmospheric pressure, and is called transthoracic pressure.

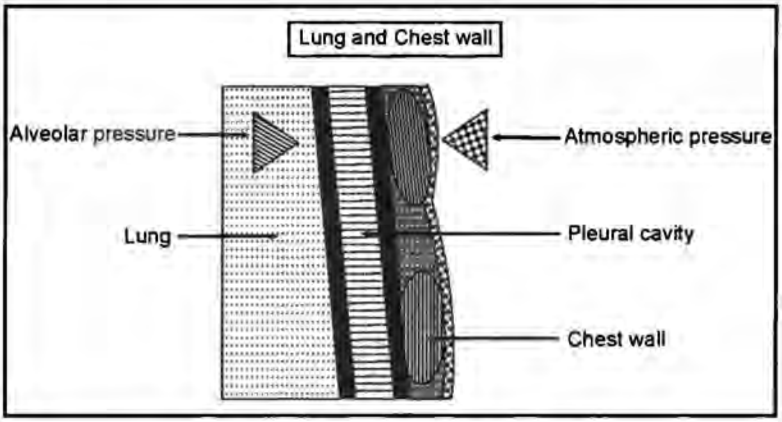


Figure 5.3: Transthoracic pressure

The compliance of the lungs is 200 ml/cm H_2O and the chest wall is 200 ml/cm H_2O. The total compliance is the combination of compliance of the lungs and the chest wall and is normally 100 ml/cm H_2O. This compliance can directly influence the volume of air that moves into the lungs by muscular force. The lungs and the thoracic wall function as a single unit, and provided the tidal volume is in the normal range, there is a linear relationship between the volume change and the pressure that produces it.

Specific Compliance

Specific compliance is the relationship of the compliance to the functional residual capacity.

$$\text{Specific compliance} = \frac{\text{Compliance (L/cmH}_2\text{O)}}{\text{Volume of lung at FRC}}$$

The specific compliance is identical in infants and adults.

Clinical Significance of Specific Compliance

- *Patient with destruction of elastic fiber:*
  When the elastic fibres are destroyed, elastic recoil is reduced. This should actually make it easy to inflate the lungs, i.e. achieve unit

volume change, with lesser pressure, but in practice, due to damage of elastic fibres, gas trapping occurs, functional residual capacity is increased, and this lung is not easy to inflate. In these types of patients accurate estimation of compliance can be better expressed by specific compliance because it is related to the lung volume.

- *Patient after pneumonectomy:*
  As the lungs and chest wall are aligned in series, the pressure required to distend the system is the sum of the pressures required to distend the lung and the chest wall together. If a person has a pneumonectomy, the remaining lung may be healthy, with normal distensibility, but it will have only half the volume as compared to both the lungs. Therefore, the change in volume for a given change in pressure will only be half the normal change. The discrepancy is eliminated if the results are expressed in terms of specific compliance.

COMPLIANCE IN THE NEWBORN

Less pressure is needed to expand the chest wall in newborns during inspiration due to:

1. Reduced elastic recoils
2. Non-ossified chest wall
3. Low total muscle mass

This explains reduced requirement of muscle relaxants for a newborn during ventilator treatment.

FACTORS AFFECTING THE COMPLIANCE

- *Posture:*
  In the supine position, the FRC is decreased, consequently the compliance is less.
- *Restriction of expansion of the chest:*
  The physical restriction of the mobility of the chest, due to abdominal distension or strapping decreases compliance.
- *IPPV:*
  IPPV reduces compliance by causing, uneven ventilation and increasing airway obstruction due to accumulation of secretion because of decreased ciliary activity if proper measures are not taken.
- *General anesthesia:*
  Supine posture, shallow breathing, increased bronchomotor tone and increased pulmonary blood flow will cause a fall in the compliance.

- *Disease states of the lung*
  - Decreased elasticity of the pulmonary fibre, e.g. pulmonary fibrosis
  - Increased intrapulmonary fluid content and decreased surfactant activity, e.g. pulmonary oedema, ARDS
  - Increased pulmonary blood volume, e.g. pulmonary congestion as in mitral stenosis, decreases lung compliance by decreasing initial lung volume.

ELASTANCE (FIG. 5.4)

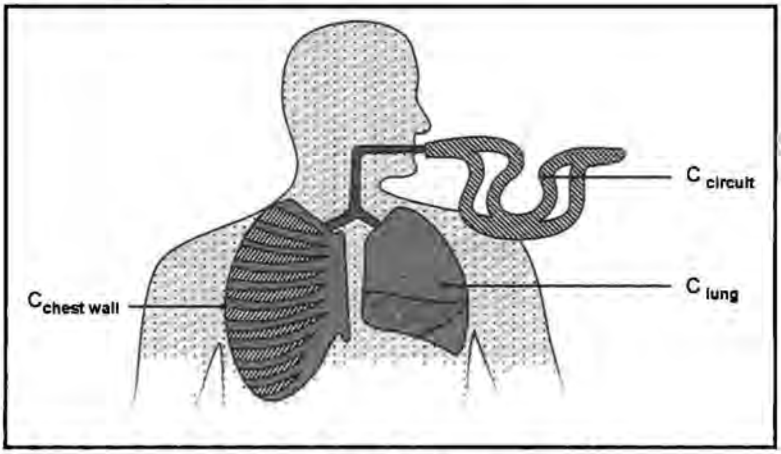


Figure 5.4: Total elastance

It is defined as the force tending to return the lung to its original size after stretching it. It is measured as pressure exerted by the recoil of the lung to reduce the volume by one unit (cmH_2O/l).

$$E_{Lung} = \Delta P_{Alv}/\Delta V$$

Total elastance (inverse of compliance) therefore, is the sum of lung and chest wall elastance.

$$1/C_{total} = 1/C_{lung} + 1/C_{chest\ wall}$$

During mechanical ventilation total elastance is made up of the circuit and the respiratory system since both are placed in parallel.

$$1/C_{total} = 1/C_{resp\text{-}System} + 1/C_{circuit}$$

Thus, during mechanical ventilation the ventilatory circuit is also expanded by the pressure applied by the ventilator.

Elastance – Elastic Recoil of the Chest Wall and Lungs

I. Elastic recoil of the chest wall
   - *Elasticity of the chest wall:*
     The elasticity of the chest wall, including the intercostal muscles, the diaphragm and also the abdomen, contributes to these forces.

- *Bony development of the cage:*
  The elastic recoil of the chest wall depends not only on elastic tissue elements but also on the bony development of the cage.

II. Elastic recoil of the lung

- *Elastic tissue of the lung:*
  The elastic fibers are present in the bronchial wall and throughout the interstitial tissue of the lungs.
- *Arrangement of the muscle fibers:*
  The arrangement of the muscle fibres of the bronchi and bronchioles are in a "geodesic" network so that on contraction, the bronchial tree reduces in diameter as well as in length.
- *Alveolar lining fluid:*
  Alveoli tend to collapse during the end of expiration due to surface tension effects of the fluid lining the alveolar walls.

SURFACTANT-ANTI-ATELECTASIS FACTOR

The surfactant, chemically dipalmitoyl lecithin, is secreted by type 2 alveolar cells and lines the alveolar surface. Surfactant is an alveolar lining substance which lowers surface tension forming an insoluble surface film at the air-alveolar interface.

Functions of the Surfactant

- *To reduce the muscular effort required for breathing*
  The primary function of the surfactant is to reduce the muscular effort required for breathing.
- *Improves the distensibility of the lungs*
  Lowering of the surface tension by the surfactant improves the distensibility of the lungs.
- *Contributes to the stability of the architecture of the lungs*
  Within the lungs, the action of the surfactant becomes more effective as the alveoli decrease in size. When alveoli decrease in size during expiration, ideally surface tension should increase, but due to the presence of the surfactant, surface tension reduces. This is most essential for the stability of alveoli, in other words, the substance at the alveolar-air interface (surfactant) is a kind of anti-atelectasis factor.

Laplace's Law

Laplace's law – states that

$$T \propto P \times r$$

Where T is tension, P is pressure and r is radius

Laplace's law explains the relationship between size of alveoli and surface tension (T). If a balloon is partially inflated, the fat portion of the balloon feels very tense but the nipple feels very flaccid. Since the two parts of the balloon are in communication with each other, the pressure inside has to be the same in both the parts but tension in the two parts is different because of the difference in radius.

Since $T \propto P \times r - P \propto T/r$

Since $P \propto T/r$, if T increases parallel with r, then T/r will be essentially constant, i.e. P will be constant.

Alveoli and Laplace's Law (Fig. 5.5)

Alveoli function in a similar manner. All alveoli do not have the same diameter. If the surface tension of the fluid lining the alveoli were the same, smaller alveoli would have higher pressure than larger alveoli and smaller alveoli would empty into larger alveoli. This process would continue till the entire lung is converted into a giant alveolus. This actually does not happen because of the surfactant in the lungs.

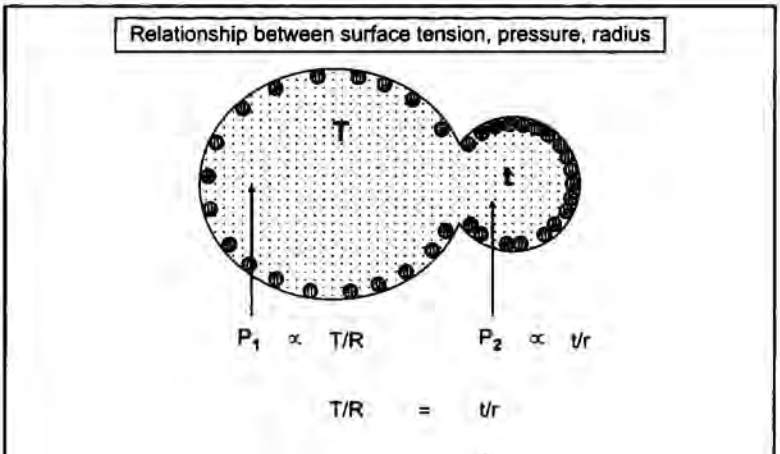


Figure 5.5: Laplace's law

The amount of surfactant secreted by an alveolus is relatively constant. In a large alveolus, the surfactant spreads over a larger surface and is therefore, less effective than in a small alveolus. In other words larger alveoli have less reduction in surface tension compared to smaller alveoli.

Therefore, the surface tension of larger alveoli is greater than that of smaller alveoli. Since $P \propto T/r$, if T increases parallel with r, T/r will be essentially constant, i.e. P will be constant.

Hence, in the lungs, the pressure in large alveoli is equal to the pressure in small alveoli. Therefore, small alveoli do not empty into large alveoli. In this way the surfactant contributes to the stability of the architecture of the lungs. This is the second function of the surfactant.

Laplace's Relationship

The amount of intra-alveolar pressure needed to counteract the tendency of bronchioles and alveoli to collapse is directly proportional to double the surface tension and inversely proportional to the radius (Laplace relationship).

The formula of Laplace states that the pressure across a curved surface is equal to twice the surface tension at the air liquid interface divided by the radius of curvature.

$$\text{Pressure} = \frac{2(\text{surface tension})}{\text{Radius}}$$

The Laplace formula implies that as the alveoli decrease in size during expiration, the pressure tending to collapse those increases and a vicious circle is established. The fact that this does not happen in reality means that the surface tension has, in some way, altered during expiration.

Causes of Low Surfactant

1. *Oxygen therapy:*
   Oxygen therapy may lead to a reduction in surfactant and hence it should be used in the lowest possible concentration and for the shortest time necessary, to achieve the desired effect.
2. *Intermittent positive pressure ventilation:*
   Intermittent positive pressure ventilation at normal pressures and volumes does not produce a fall in surfactant but when abnormally high pressures are used, there is a marked reduction in surfactant concentration with consequent atelectasis.
3. *Deficiency in pulmonary surfactant:*
   In RDS, deficiency in pulmonary surfactant will result in the decreased stability of the bronchioles and alveoli, and thus result in a tendency to collapse.

CLINICAL SIGNIFICANCE OF ABNORMAL COMPLIANCE

These abnormalities are often contributing factors to the need for mechanical ventilation:

- Abnormally low or high lung compliance impairs the patient's ability to maintain an efficient gas exchange.
- Low compliance typically makes lung expansion difficult and high compliance induces incomplete exhalation and CO_2 elimination.

Low Compliance—High Elastance (Fig. 5.6)

If small volume change is produced after applying a relatively large amount of airway pressure, lung is stiff or noncompliant, poorly distensible, and resists expansion. Low compliance means that the volume change is less per unit pressure change. Reduced compliance is also referred to as a restrictive ventilation disorder.

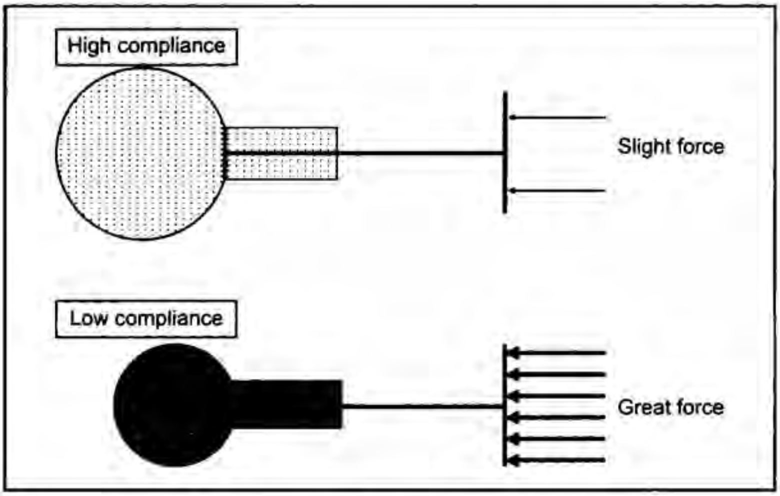


Figure 5.6 Lung compliance under artificial ventilation

Anaesthetic Significance of Low Compliance

Low compliance measurements are usually related to conditions that reduce the patient's functional residual capacity. Patients with noncompliant lungs often have a restrictive lung disease, low lung volumes, low minute ventilation. This condition may be compensated for by an increased respiratory rate. The work of breathing is high when compliance is low. In many clinical situations (e.g. atelectasis), low lung compliance is responsible for refractory hypoxaemia.

Clinical Conditions with Reduced Compliance

I. *Decreased Compliance of Lungs-Restrictive Lung Disease*

1. *Parenchymal alteration*
   - Altered surface tension with fluid in the alveoli
     Pulmonary oedema, ARDS
   - Decreased elasticity of the pulmonary fibre
     Pulmonary fibrosis
     Pulmonary oedema, ARDS

   - Inflammatory conditions
     Bronchopneumonia
   - Increased pulmonary vascular congestion
     Mitral stenosis.
2. *Deficient Surfactant*
   - ARDS, Atelectasis
   - Aspiration.
3. *With low lung volumes-Diaphragmatic restriction*
   - Abdominal distension
   - Increased abdominal pressure e.g. peritoneal dialysis
   - Postlaparotomy
   - Congenital pulmonary hypoplasia
   - Diaphragmatic hernia.
4. *Intrinsic lung restriction and expansion*
   Congenital emphysematous lung Emphysema – destruction of elastic fibers.

B. *Decreased Chest Compliance*

1. *Extrinsic lung restriction—Restrictive pleural disease*
   - Pleural collection of air or fluid
   - Fibrosis
   - Tension pneumothorax
2. *Increased intercostal muscle tone*
   - Upper motor neuron disease
   - Drugs
3. *Restrictive chest diseases*
   - Deformation- kyphosis, Scoliosis, or both
   - Ankylosis
   - Restrictive bandages

High Compliance–Low Elastance (Fig. 5.6)

If a large change in volume is achieved by applying a relatively small amount of airway pressure, the lung is easily distensible and is said to be highly compliant.

High compliance means that the volume change is large per unit pressure change.

Clinical Presentation of High Compliance

1. High compliance measurements are usually related to conditions that increase the patients' functional residual capacity. Emphysema is an example of a condition with high compliance where the gas exchange

process is impaired. In emphysema the recoil of the lung is reduced due to the loss of pulmonary parenchyma. This condition is due to chronic air trapping, destruction of lung tissue, enlargement of terminal and respiratory bronchioles.

2. Patients with extremely compliant lungs often have: Obstructive lung disease, e.g. asthma, airflow obstruction, incomplete exhalation, poor gas exchange.
3. In extreme high compliance situations, exhalation is often incomplete due to lack of elastic recoil by the lungs.

COMPLIANCE AND WORK OF BREATHING

1. Since compliance is inversely related to pressure change (ΔP - Work of breathing, ΔV = Lung expansion producing tidal volume), a decrease in compliance means an increase in the work of breathing.
2. In a clinical setting, hypoventilation usually results when a patient is unable to compensate for the decrease in compliance by increasing and maintaining a higher level of work of breathing.
3. Atelectasis is one of the frequent causes of increased work of breathing.
4. In low compliance situations such as ARDS, fibrosis, and kyphoscoliosis, the decrease in minute ventilation is characterized by small tidal volumes and fast respiratory rates a sign of volume restriction.

Effects on Ventilation and Oxygenation

Abnormal compliance impairs the gas exchange mechanism. When an abnormally low or high compliance is not corrected and prolonged, muscle fatigue may occur leading to the development of ventilatory and oxygenation failure.

1. Ventilatory failure develops when the patient's minute ventilation cannot keep up with the CO_2 production.
2. Oxygenation failure usually follows when the pulmonary system cannot supply the oxygen needed for metabolism.

BABY LUNG (FIG. 5.7)

It is important to emphasize that the measurement of both R and C only reflects the condition of the part of the lung that is ventilated. Thus, a low C or high R can be caused by a small lung, i.e. the "baby lung" in ARDS and the other types of "baby-lung" in severe asthma, where most of the lung volume is trapped behind closed obstructed airways.

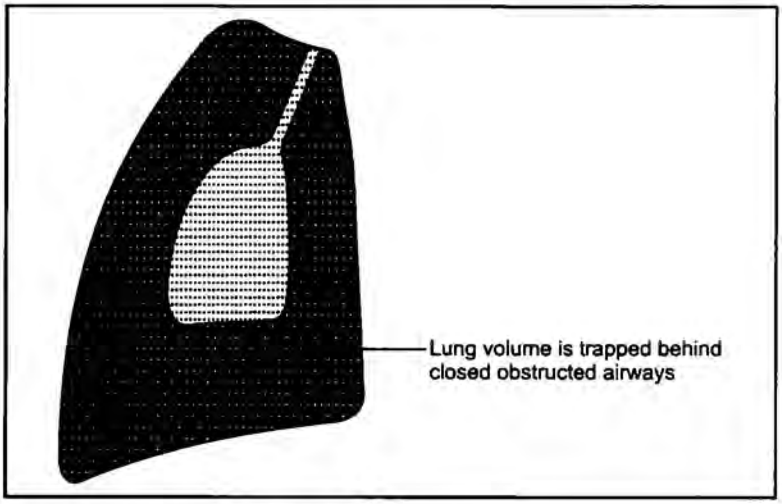


Figure 5.7: Baby lung

CHAPTER

6

Resistance

DEFINITION

Resistance is the result of friction between moving parts. Liquids or gases flowing through tubular structures have to overcome the resistance due to friction between the molecules of the substance flowing and the wall of the tube. A certain pressure is required to deliver the gas through the airways to the alveoli. This pressure difference between the mouth and the alveoli, needed to move the required volume of air across the airways, is determined by the resistance offered by the airways.

The airway resistance follows a mathematical relationship similar to that of electrical resistance, as given by Ohm's law. Thus, in the respiratory system airway resistance (R) is given by the equation:

$$R = \frac{\Delta P}{\dot{V}}$$

ΔP is the difference in pressure (transairway) between the two ends of the tube (alveolar pressure and pressure at the mouth, i.e. atmospheric pressure) and $\dot{V}$ is the rate of the flow.

Volume = Flow rate × time

Lit = (lit/sec) × sec

$$\text{Flow rate (lit/sec)} = \frac{\text{Volume(Litre)}}{\text{Time (Second)}}$$

Flow rate is volume per unit time.

$$\text{Resistance} = \frac{\text{Propelling pressure (cmH}_2\text{O)}}{\text{Gas flow (rate of flow) (lit/sec)}} = \text{cmH}_2\text{O/(lit/se)}$$

In other words, resistance is unit pressure change per unit rate of flow.

CLASSIFICATION OF FLOW

The flow can be classified as:

1. *Laminar flow (Figs 6.1 and 6.2):*
   When the flow of gas along a tube is smooth and regular it is called laminar flow. Laminar flow is silent and occurs mostly in the airways below the main bronchi. The flow of gases occurs in the form of concentric cylinders sliding one over the other, with the central cylinder moving faster than the peripheral cylinder.

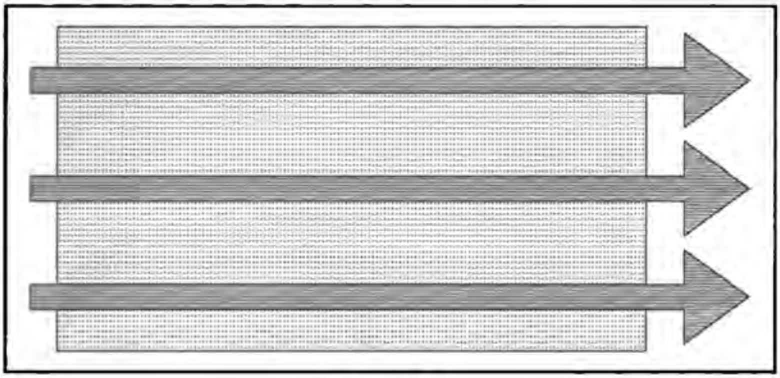
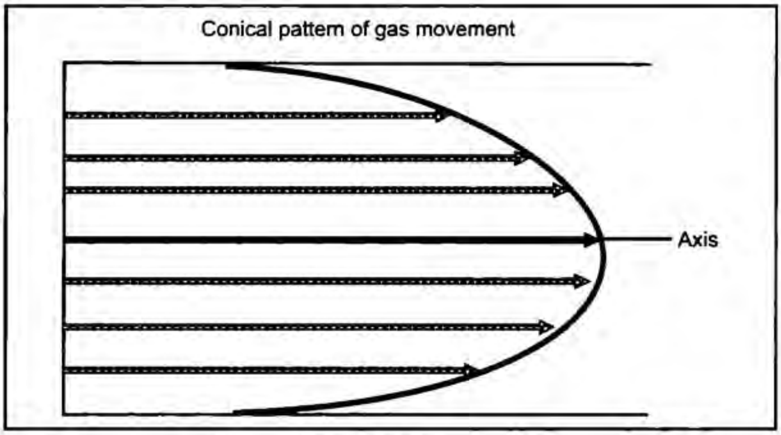


Figure 6.1



Figures 6.1 and 6.2: Laminar flow

The conical pattern of gas movement in a laminar flow has the advantage of having more surface area for the exchange of gases. This may particularly explain the survival of patients on a very small tidal volume of about 100 ml. It may also partly explain the efficacy of very low tidal volumes in high frequency ventilation (HFV).

2. *Turbulent flow (Fig. 6.3):*
   As the rate of flow increases, a critical velocity is reached after which the flow becomes turbulent. When the flow in the airway exceeds a critical velocity (defined as Reynolds's number more than 2000) then the flow becomes turbulent.

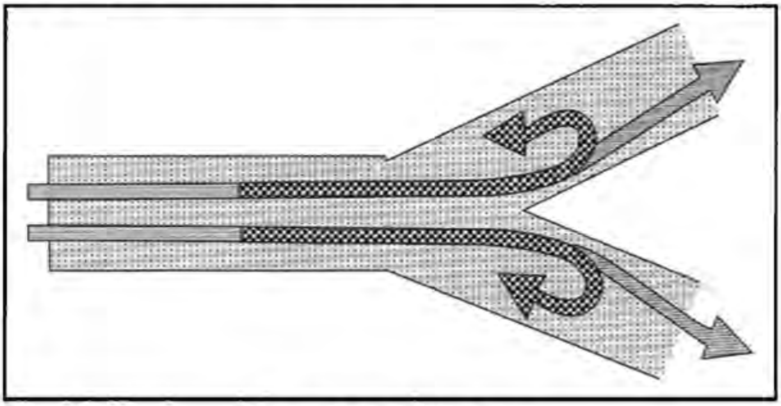


Figure 6.3: Turbulent flow

   But a turbulent flow is also more likely when the diameter decreases or the direction of the tube is changing or when branching occurs. Turbulent flows are usually audible. The flow in the trachea is turbulent.

3. *Orificial flow:*
   This occurs at constrictions like the larynx. Here, the flow rate is inversely proportional to the density.

SITES OF AIRWAY RESISTANCE

The airways divide repeatedly like branches of a tree. Each successive branching has been numbered and is referred to as a generation. The division goes on till about the 20th generation. Airway resistance resides mainly in medium sized airways i.e. airways up to the 7th generation. About 9/10th of the pressure required for driving the gas in and out of the lungs is used for overcoming resistance in the medium bronchi.

The wider, large airways offer less resistance. The small airways are so many that their total cross sectional area is much greater at the bronchiolar level as compared to that found centrally. Hence, small airways also do not offer much resistance. Only 1/10th of the pressure is required for driving the gas through the bronchioles.

Alteration of Resistance during the Respiratory Cycle

As explained above, the tracheobronchial tree is not a rigid system of pipes. As the bronchi loose their cartilaginous support by the 11th to the 13th generation, effective forces can distend or narrow these airways more than the cartilage containing parts of the bronchial system. The lumina of the cartilage free bronchioles are kept open mainly by means of the elastic recoil of the surrounding pulmonary tissue.

Factors Essential for Keeping the Small Airways Open

1. Elastic recoil of the lungs
2. The intrapleural pressure—The pressure exerted on the total lungs including the airways.

Bronchial Lumen Variation with Phases of Respiration (Fig. 6.4)

During inspiration, elongation of the elastic pulmonary fibres increases the elastic retraction pressure (comparable with a coiled spring). The bronchioles are stretched by the stronger radial pull and the bronchial flow resistance falls. With expiration, due to the elastic recoil, the bronchi become narrower and the flow resistance increases.

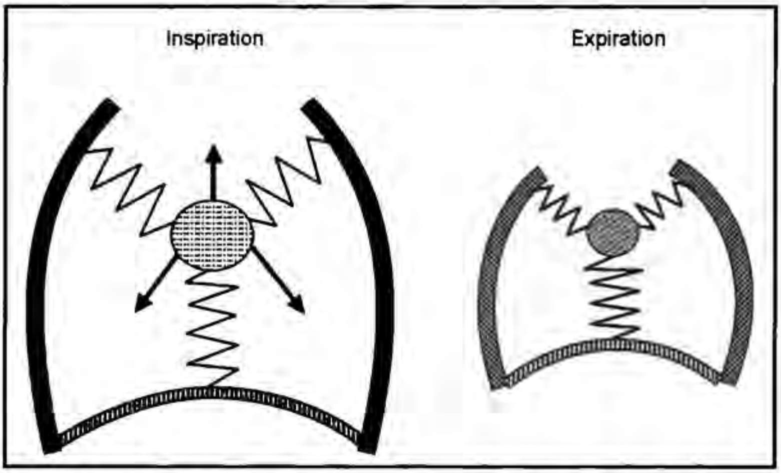


Figure 6.4: Bronchial lumen variation with phases of respiration

These cyclical changes of flow resistance explain why the expiratory phase is always slightly longer than the inspiratory phase. That is why expiration, rather than inspiration, always plays a major role in obstructive ventilatory disorders. Accordingly expiration becomes prolonged, more difficult and expiratory obstructive signs such as wheezing or rhonchi can be auscultated over the lungs.

Variation of Pressure in the Alveoli

During inflation of the lungs the pressure at the mouth must be greater than the pressure in the alveoli; otherwise no gas would flow into the alveoli. Conversely, during the expiratory phase, the pressure in the alveoli must be greater, than mouth so that flow would flow to exterior. However there is at least an instant of time between the two phases when the pressure is the same throughout the respiratory tract and there is no gas flow in either direction, e.g. at end of inspiration and expiration.

Alveolar Pressure Range (Fig. 6.5)

The alveolar pressure (P_{alv}) is the sum of the intrapleural pressure (P_{ple}) and the elastic recoil pressure (elastance) of the lungs.

$$P_{alv} = P_{ple} + P_{elastance}$$

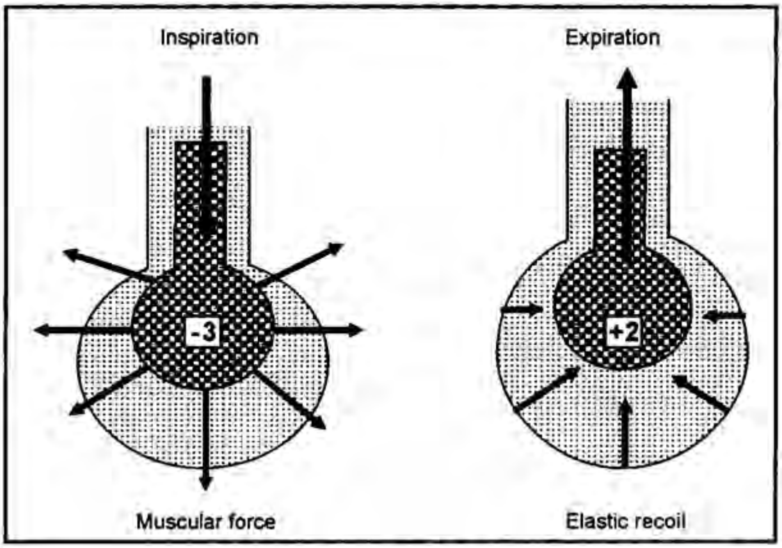


Figure 6.5: Alveolar pressure range

The alveolar pressure range is the difference between the pressures in the alveoli at the end of inspiration to the end of expiration. The end inspiratory and end expiratory pressures in the alveoli are primarily influenced by the pressure at the mouth end (atmospheric pressure when the patient is breathing spontaneously) and the pressure difference across the airways.

RWAY RESISTANCE

Airway resistance is defined as the change in transairway pressure proximal airway pressure i.e. atmospheric pressure-alveolar pressure) required to produce a unit flow of gas through the airways of the lung. mH$_2$O/lit/sec.)

Resistance and Compliance (Figs 6.6 and 6.7)

In addition to the pressure required to overcome the elasticity of the respiratory system, pressure is needed to force a gas through the airways at a constant flow rate (airway resistance) and exceed the viscous resistance of the lung tissue (tissue resistance).

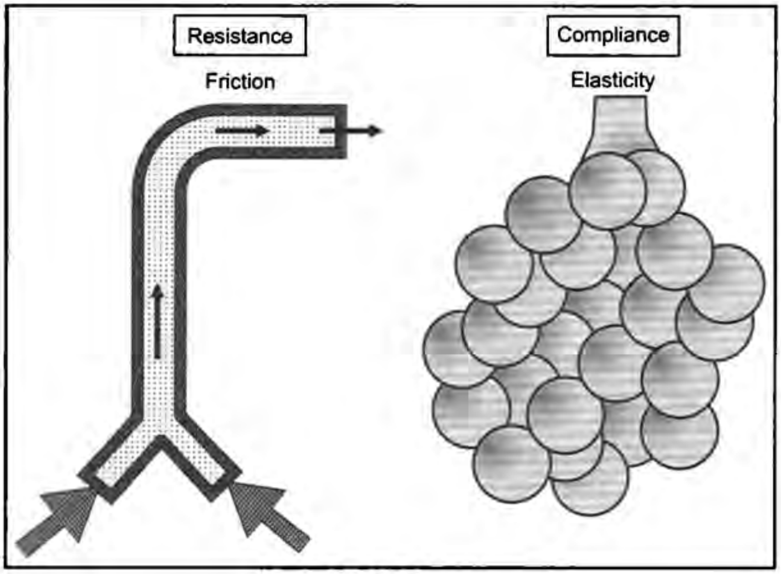
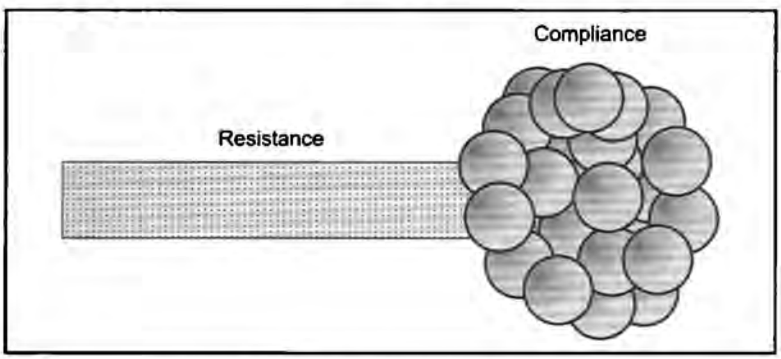


Figure 6.6



Figures 6.6 and 6.7: Resistance and compliance

$$\text{During expiration } R = \frac{\text{Alveolarpressure} - \text{Proximal airway pressu re}}{\text{Rate of flow}}$$

$$\text{During expiration } R = \frac{\text{Change in transairway pressure}}{\text{Rate of flow}}$$

Normal airway resistance is between 0.6 and 2.4 cm H_2O/L/sec at a flow rate of 30L/min (0.5 L/sec.). The normal airway resistance in healthy adults is 1.6 cmH_2O/lit/sec.

Factors Determining Airway Resistance

A large number of physiological and pathological factors influence airway resistance.

- Properties of the airway and types of gas flow
- Lung volume
- Bronchial muscle tone
- Dust and smoke.

Properties of an Airway

Radius, length and branching and surface characteristics also play an important role in determing airway resistance.

Gas flow

Rate of flow: Resistance to a great extent is dependent on the flow, increasing as the flow rate increases.

Types of flow and diameter of the tube (Fig. 6.8)

1. *Laminar flow:* The gas molecules flow linearly along the airway resulting in lower resistance. The determinants of airway resistance during laminar flow are the viscosity of gas and the length of the airway. Resistance to airflow is inversely proportional to the fourth power of the airway radius (r) i.e. resistance increases sixteen fold if the airway diameter is reduced by half, which increases the work of breathing.

   The flow rate in laminar flow is governed by Hagen-Poiseuille's law i.e. the flow rate is directly proportional to the pressure difference across the tube and to the fourth power of the diameter of the tube. Decrease in the diameter of the airway by half would therefore decrease the flow rate to $1/16^{th}$. The flow rate is also inversely proportional to the viscosity of the gas.

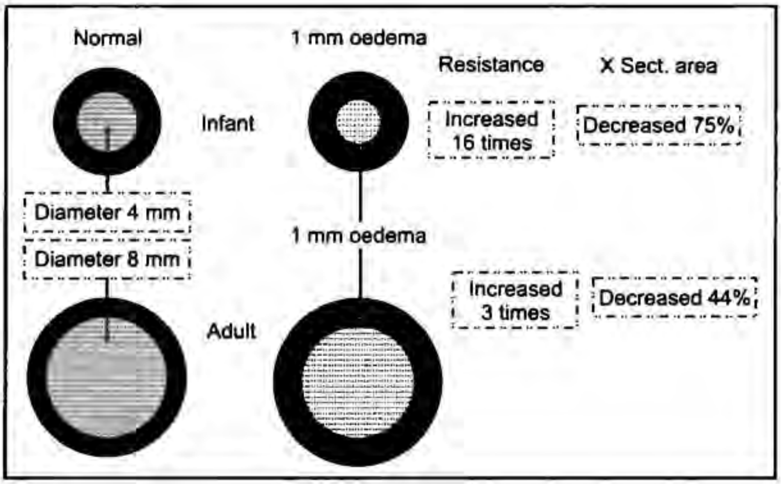


Figure 6.8: Effects of oedema on airway resistance in the infant versus the adult

Hagen Poiseuille's law $R \propto \dfrac{L}{r^4} \propto \dfrac{\Delta P}{\dot{V}}$

L & r are the length and radius of the airway, respectively.

Poiseuille's law $\Delta P \propto \dfrac{\dot{V}}{r^4}$

$$\dot{V} \propto \Delta P \times r^4$$

2. *Turbulent flow:* Eddies occur, caused by higher flow rates, uneven surfaces and branching, resulting in higher resistance.

   Hagen Poiseuille's law — $R \propto \dfrac{L}{r^5}$

In turbulent flow, the pressure gradient is proportional to the fifth power of the radius i.e. resistance to airflow is inversely proportional to the fifth power of the radius. During crying the airflow becomes turbulent and airway resistance increases markedly, increasing the work of breathing. Due to this reason, the infant or child with an airway obstruction should be kept as calm and quiet as possible to prevent the generation of a turbulent airflow.

Lung Volume

During inspiration, small airways get stretched and widened. This is partly due to the increase in the lung volume and partly due to the negative pressure in the alveoli. Hence, airway resistance is lower during inspiration. Airway resistance is least at lung volumes near the FRC.

Conversely during expiration, the lung volume gets smaller and the positive alveolar pressure compresses the airways. Some small airways might even collapse and close completely during forceful expiration. The expiratory flow rate cannot be increased beyond a certain limit in spite of increasing muscular effort during expiration.

Bronchial Muscle Tone

Contraction of the bronchial musculature reduces the radius of bronchi and therefore increases the airway resistance. Bronchial musculature is under autonomic control. Sympathetic stimulation relaxes the smooth bronchial muscle whereas parasympathetic stimulation induces its contraction.

Various diseases affecting Bronchial muscle tone

Patients find expiration much more difficult than inspiration during an attack of bronchial asthma and rhonchi are always expiratory. In contrast, if rhonchi are due to an obstruction of the large airways by mucosal swelling or viscid secretions (as in bronchitis), they are heard during both inspiration and expiration.

Resistance is also increased if any type of swelling obstructs the airways from within or due to pressure on the airways from outside.

- Inside the airway – e.g. retained secretions
- In the wall of the airway – neoplasm of the bronchial muscle structure.
- Outside the airway – tumors surrounding and compressing the airway.

Dust and Smoke

Dust and smoke particles add to the friction associated with the airflow during breathing. Therefore, airway resistance increases in a polluted atmosphere and during smoking.

Features of Increased Airway Resistance

The characteristic diagnostic feature of increased airway resistance is typically slower expiration so that the fraction of vital capacity expired in one second (EFV_1) is reduced. This has a number of potential consequences:

1. *Decreased respiratory rate:*
   Since expiration needs more time, the respiratory frequency may be reduced. However, this cannot go beyond a certain limit because it impairs alveolar ventilation.
2. *Use of accessory muscles:*
   Expiration is quickened by employing expiratory muscles. This has limits because it makes the patient tired and the increased intrathoracic pressure compresses the airways, further increasing the airway resistance.

Dynamic Airway Compression (Fig. 6.9)

Even during forced expiration an increase of intrapleural pressure more than + 40 mbar can cause dynamic compression of the small airways.

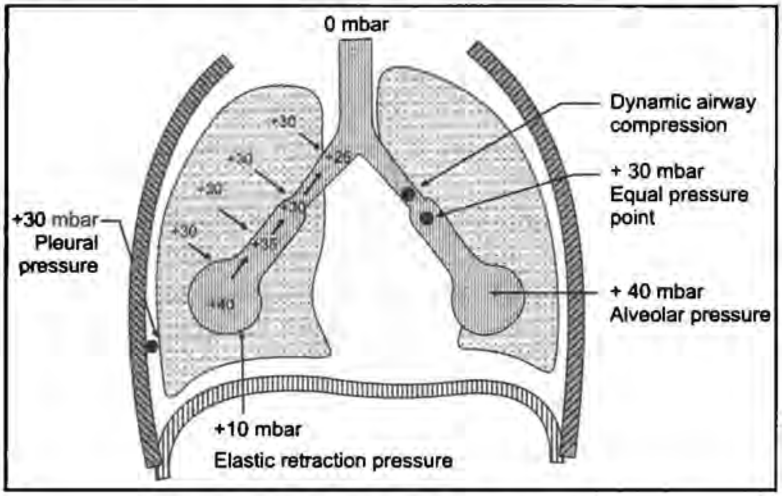

0 mbar
Dynamic airway compression
+ 30 mbar Equal pressure point
+30 mbar Pleural pressure
+ 40 mbar Alveolar pressure
+10 mbar Elastic retraction pressure
+30
+30
+30
+30
+25
+30
+35
+40

Figure 6.9: Dynamic airway compression

This results in extreme narrowing or even closure of the bronchiole and occurs when the intrapleural pressure is considerably larger than intraluminal pressure.

Energy Expenditure for Airway Resistance

The respiratory muscles use most of the energy in overcoming elastic recoil of the lungs and the chest wall. In a normal man, breathing at 12 -15 times a minute, more than two thirds of the energy is spent in overcoming the elastic forces. However, the situation is reversed if the rate is increased to 50 times per minute, then more than two thirds of the work is spent in overcoming airflow resistance.

Clinical Implications of Airway Resistance

In mechanical ventilation, the degree of airway resistance is primarily affected by length, size and patency of the endotracheal tube and ventilatory circuit. The minimal flow resistance in large bore endotracheal tube is 3 to 7 cm H_2O/L/sec, so non-pulmonary resistive elements occupy a considerable fraction of the total inspiratory resistance.

Internal Diameter of the ETT

Resistance is higher in intubated patients, depending on the size of the ETT. The internal diameter of the ETT is a major contributor to increased airway resistance. Therefore, during intubation, the largest appropriate size ET tube must be used so that the airway resistance contributed by the ET tube may be minimized.

Patency of ETT

Once the ET tube is in place, its patency must be maintained as secretions retained inside the ET tube greatly increase airway resistance.

Length of ETT

Airway resistance varies directly with the length and inversely with the diameter of the ETT. In a clinical setting, the ETT is sometimes cut shorter for ease in airway management, reduction of mechanical dead space and reduction of airway resistance.

Ventilatory Circuit (Fig. 6.10)

During mechanical ventilation the resistance of the endotracheal tube (R_2) is often greater than that of the respiratory system (R_1). Besides the ETT tube, the ventilator circuit may also impose mechanical resistance to the airflow and contribute to total airway resistance. This is particularly important when there is a significant amount of water in the ventilator circuit due to condensation.

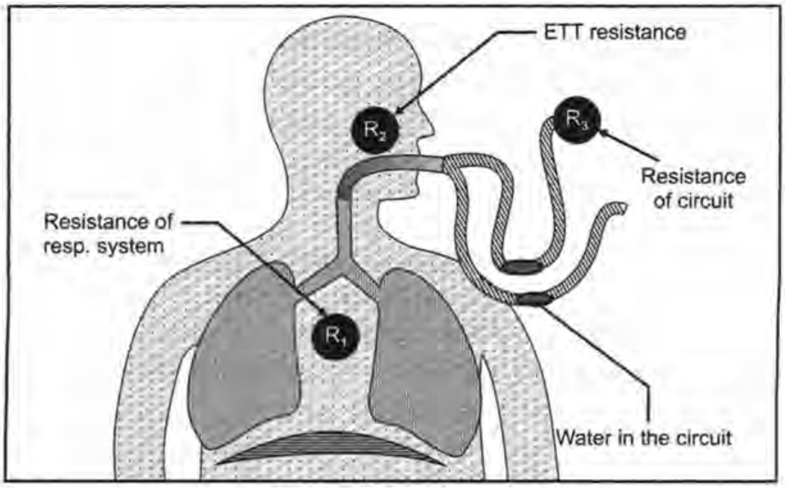


Figure 6.10: Total resistance

The three resistances are placed in series and are therefore additive:

$$R_{\text{total}} = R_{1\ \text{resp. system}} + R_{2\ \text{tube}} + R_{3\ \text{Circuit}}$$

Airway, lung tissue and circuit resistance are together defined as total pulmonary resistance.

Airway Resistance and Infant

In an infant the airway resistance is equally distributed between the upper and lower airways. Airway resistance values for normal, spontaneously breathing newborn infants are generally in the range of 20-30 cm H_2O / l / sec. Values of total pulmonary resistance for intubated infants range from approximately 50-150 cm H_2O/l/sec.

Resistance of the Nasal Passages in Newborn

1. The resistance of the nasal passages in the spontaneously breathing newborn is as much as 40% of the total airway resistance and the rest is "pulmonary".
2. Neonates are obligatory nose breathers and use of a nasogastric tube, nasal prongs or an endotracheal tube can increase nasal resistance significantly.

Tissue Resistance

There is also viscous tissue resistance to expansion of the lungs. This arises to a small extent in the lung tissue itself, but mostly in the tissues of the chest wall as a result of friction between the tissues.

Airway Resistance and Work of Breathing

$$R_{aw} = \frac{\Delta P}{\dot{V}} = \frac{\text{Pressure change}}{\text{Flow}}$$

The pressure change in the equation can be treated as the amount of work of breathing imposed on the patient. Since airway resistance is directly related to pressure changes (the work of breathing), an increase in airway resistance means an increase in the work of breathing. In a clinical setting, airway obstruction is one of the most frequent causes of increased work of breathing. If pressure change (work of breathing) in the equation above is held constant, an increase in airway resistance will cause a decrease in the flow and subsequently a decrease in minute ventilation. This is because airway resistance and flow in the equation are inversely related.

Effects on Ventilation and Oxygenation

The work of breathing imposed on a patient is increased when airway resistance is high. This creates a detrimental effect on the patient's ventilatory and oxygenation status. If an abnormally high airway resistance is sustained over a long time, fatigue of the respiratory muscles occurs, leading to ventilatory and oxygenation failure. In a clinical setting, hypoventilation may result if the patient is unable to overcome the airway resistance by increasing the work of breathing.

CHAPTER

7

Shunt

Excess perfusion ("wasted" perfusion) in relation to ventilation refers to shunt. This is in contrast to dead space ventilation ("wasted" ventilation). Blood, passing through the alveolus which is not adequately ventilated but normally perfused, will not be adequately oxygenated because it has not come in contact with ventilated and oxygenated alveoli. It means blood has flown from the right heart to the left heart without being adequately oxygenated. This mechanism is responsible for intrapulmonary right to left shunt (Fig. 7.1).

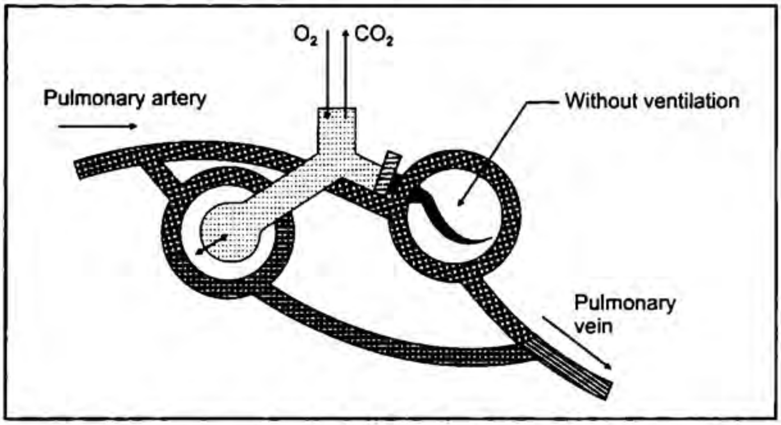


Figure 7.1: Intrapulmonary right to left shunt

In cases of increased intrapulmonary right to left shunt the arterial blood does not contain sufficient oxygen and central cyanosis develop. Intrapulmonary shunting gives rise to refractory hypoxemia i.e. low oxygen levels in the blood that responds very poorly to oxygen therapy alone. Administration of a high inspired oxygen concentration can partly compensate for an increased intrapulmonary right to left shunt depending on shunt fraction. A normal value of intrapulmonary right to left shunt is 3-5%.

MICROATELECTASIS

Every single collapsed (atelectatic) alveolus causes a right to left shunt due to relative overperfusion. Such a collapsed alveolus can be seen neither microscopically nor radiographically. They are, therefore, referred to as microatelectasis. Such microatelectasis may be distributed in thousands throughout the lungs so that enormous right to left shunt can exist, even though nothing pathological can be recognized radiographically.

VENOUS ADMIXTURE

The blood returning to the left side of the heart can be considered to be made up of two parts.

1. *No V_A/Q mismatch*
   Blood which has passed through ideal alveoli, that is alveoli with perfect ventilation and perfusion, has a PaO_2 equal to ideal alveolar PAO_2.
2. *Mixed venous blood*
   The oxygen and carbon dioxide contents of venous blood vary depending on which organ or part of the body the blood has come from. By the time the venous blood has reached the pulmonary artery mixing has already occurred, so that a blood sample taken from the pulmonary artery consists of mixed venous blood. The less than fully oxygenated blood which is responsible for the venous admixture effect can be divided into:
   a. *Low V_A/Q ratio*
      Blood which has picked up some oxygen but less than fully oxygenated blood i.e. blood that has passed through relatively underventilated or overperfused zones of the lung.
   b. *True shunt or Anatomical shunt*

Causes of right to left shunt (hypoxaemia due to venous mixing) are as follows:

- *True shunt or anatomical shunt (Fig. 7.2):*
  Blood which has passed from the right to the left side of the circulation and picked up no oxygen in the lungs. These shunts are

present in the normal person due to the besian veins, bronchial veins and pleural veins. The normal value is 2% of cardiac output.

- *Functional shunt–right to left shunt (Fig. 7.2):*
  Right to left shunt is an extreme example of mismatch, which in alveoli ventilation is absent but perfusion is preserved.

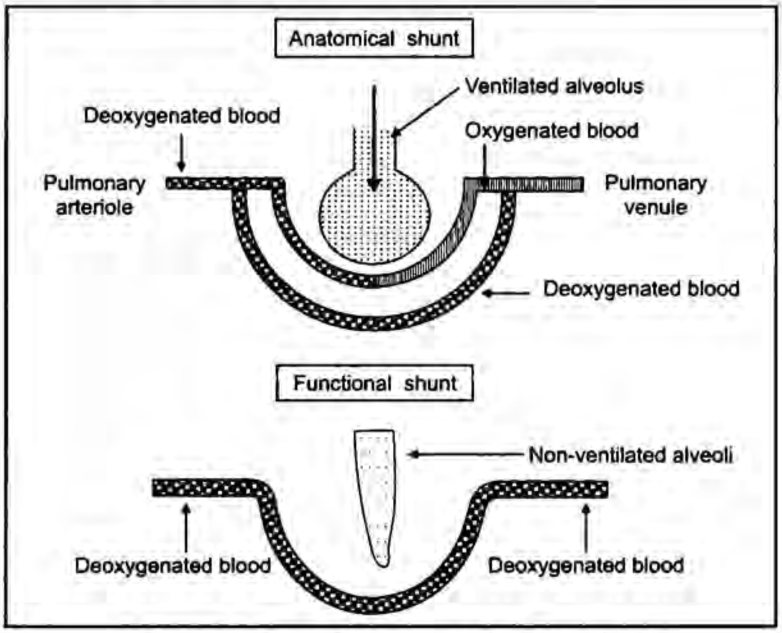


Figure 7.2: Anatomical shunt and functional shunt

Blood has passed through collapsed (underventilated) zones of the lungs and inspired oxygen has no contact with perfused blood, e. g. atelectasis, pulmonary infarction, pneumonia, pulmonary oedema or pulmonary effusion. In these patients V/Q ratio is zero and increased concentration of supplemental oxygen will not increase arterial oxygen tension because inspired air has no access to the blood perfusing the underventilated alveoli.

- *Pathological shunt:*
  Those forms of anatomical shunt are not present in the normal subject, e.g. congenital heart disease with right to left shunting.

Effects of Venous Admixture

Due to venous admixture there is an increase in arterial CO_2 content and also reduction in arterial O_2 content which is similar in magnitude. In a normal subject both quantities are very small, i.e. about 0.3 volume %. Because of the shapes of the CO_2 and O_2 dissociation curves, this small increase in CO_2 content is reflected by only a small increase in $PaCO_2$ (less than 1 mm Hg), though the small reduction in O_2 content is reflected by a large reduction in PaO_2 (around 15 mm Hg).

Best Indicator of Venous Admixture-arterial PaO_2

Due to the above reasons, the arterial PaO_2 is always taken as the best indicator of venous admixture. Though V/Q mismatch, shunt, alveolar hypoventilation and diffusion defect will give rise to hypoxia, right to left shunt is the only cause of refractory hypoxaemia. The best way to differentiate between true shunt and ventilation/perfusion mismatch is to give the subject 100% O_2 to inhale. If hypoxemia is likely to be due to shunt and if there is only a relatively insignificant rise in arterial PaO_2 it signifies shunt fraction is above 30%.

Disorders that Produce a Right to Left Shunt

- Alveoli filled with thin fluid – Cardiogenic pulmonary oedema
- Alveoli filled with exudates – ARDS, lobar pneumonia
- Closure of alveoli – atelectasis
- Reflex closure of alveoli (hypoxic pulmonary vasoconstriction response) – pulmonary emboli

Venous admixture which is normally up to 3-5% of the cardiac output may be calculated using the shunt equation and the alveolar equation.

SHUNT EQUATION

Cardiac output = Pulmonary capillary blood flow + blood through shunt

$$Q_T = Q_C + Q_S$$

The ratio of shunted cardiac output Q_S to total cardiac output Q_T is defined as the intrapulmonary right to left shunt.

$$Q_S : Q_T$$

Estimation of Shunt %

The shunt percent can be calculated or estimated by many methods, ranging from simple (less accurate) to complex (more accurate).

The clinical uses of two common calculations are discussed here:

1. Classic shunt equation
2. Estimated/Modified shunt equation

Classic Shunt Equation

The classic physiologic shunt equation requires an arterial blood sample and a mixed venous blood sample from the pulmonary artery (through pulmonary artery catheter). It is the most accurate among all shunt equations.

$$\frac{Q_S}{Q_T} = \frac{(C_CO_2 - CaO_2)}{(C_CO_2 - C\overline{v}O_2)}$$

Q_S/Q_T: Shunt fraction - Physiologic shunt to total perfusion (cardiac output) ratio in vol %

C_CO_2: Oxygen content (in vol %) of pulmonary end-capillary blood

CaO_2: Oxygen content (in vol %) of arterial blood

$C\overline{v}O_2$: Oxygen content (in vol %) of mixed venous blood.

Estimated or Modified Shunt Equation

The modified physiologic shunt equation requires only an arterial blood sample (arterial blood gases). It does not require a mixed venous blood sample from the pulmonary artery and therefore it is noninvasive and simple to perform. This estimated method is more meaningful when serial measurements are used to establish a trend. In healthy individuals, the physiologic shunt approximates the anatomic shunt and is less than 5%.

Forms of Modified Shunt Equation

Two forms of this equation are possible:

1. *Non-critical patients*
    - Spontaneously breathing
    - Moderate level of F_iO_2
    - Moderate level of continuous positive airway pressure

$$\frac{Q_S}{Q_T} = \frac{(C_CO_2 - CaO_2)}{[5 + (C_CO_2 - CaO_2)}$$

For non-critically ill patients, the normal physiological shunt is less than 10%.

2. *Critically ill patients*
    - Mechanical ventilation
    - High F_iO_2
    - High level of positive end-expiratory pressure

$$\frac{Q_S}{Q_T} = \frac{(C_CO_2 - CaO_2)}{[3.5 + (C_CO_2 - CaO_2)]}$$

In severe disease states, the physiologic shunt may be greater than 30%.

Interpretation of shunt percent

Physiologic shunt	*Interpretation*
< 3–10 %	Normal
10–20 %	Mild shunt
20–30 %	Significant shunt
> 30 %	Critical and severe shunt

Clinical significance of shunt percentage

1. When the shunt percentage is too high, oxygenation becomes an extremely difficult task for the cardiopulmonary system to support.
2. Over a period of time, the respiratory muscles will fatigue resulting in ventilatory failure.
3. This is usually followed by oxygenation failure if ventilatory interventions are not done. To improve refractory hypoxaemia, PEEP is the therapy of choice.

Analysis of Iso-shunt Diagram (Fig. 7.3)

The arterial oxygen partial pressure (PaO_2) is plotted against the inspiratory oxygen concentration (FiO_2). Analysis of this iso-shunt diagram shows that when the shunt fraction exceeds 30% of the cardiac

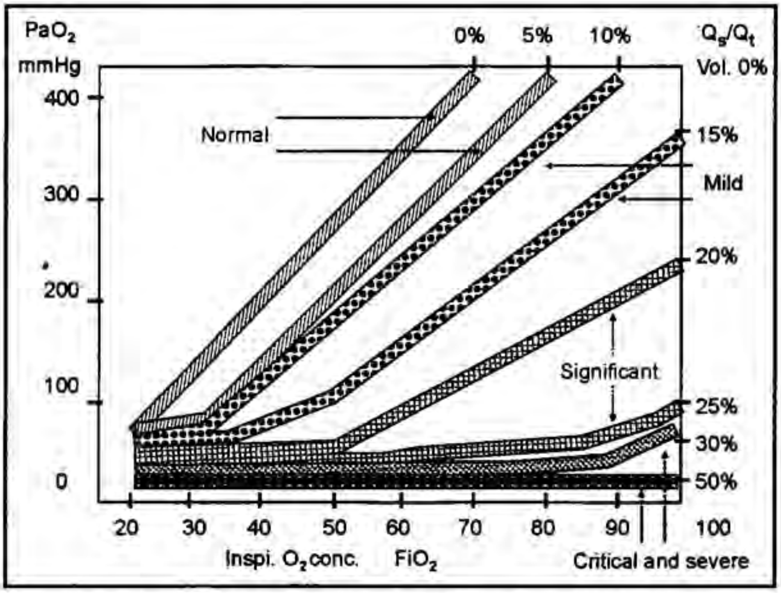


Figure 7.3: Analysis of iso-shunt-diagram (For colour version *see* Plate 1)

output, simultaneous increase in the inspiratory oxygen concentration has little effect on the partial pressure of arterial oxygen PaO_2 and it will not increase PaO_2.

The size of the intrapulmonary right to left shunt depends on the size of the collapsed lung area. To improve oxygenation, due to increased shunt fraction PEEP is the only criteria in mechanical ventilation as sigh breathing is in spontaneously breathing patients.

CHAPTER

8

Distribution of Ventilation and Perfusion

WAYS TO EXPRESS V/Q RATIO

∵ Q ratio can be expressed in many ways. They are as follows:

- It is the relation between air flow in the alveoli and blood flow in the pulmonary capillaries (Fig. 8.1).

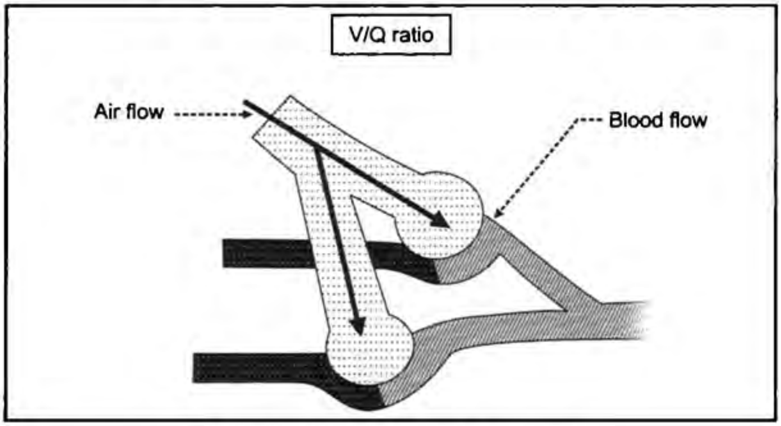


Figure 8.1: Ventilation perfusion ratio

- It is the ratio between the amount of ventilation and the amount of perfusion in the lungs.
- It is the ratio between alveolar ventilation and pulmonary blood flow i.e. cardiac output Q.

GAS EXCHANGE UNIT (FIG. 8.2)

The gas exchange unit is composed of an alveolus and its associated pulmonary capillary. This theoretical respiratory unit can exist in one of four relationships:

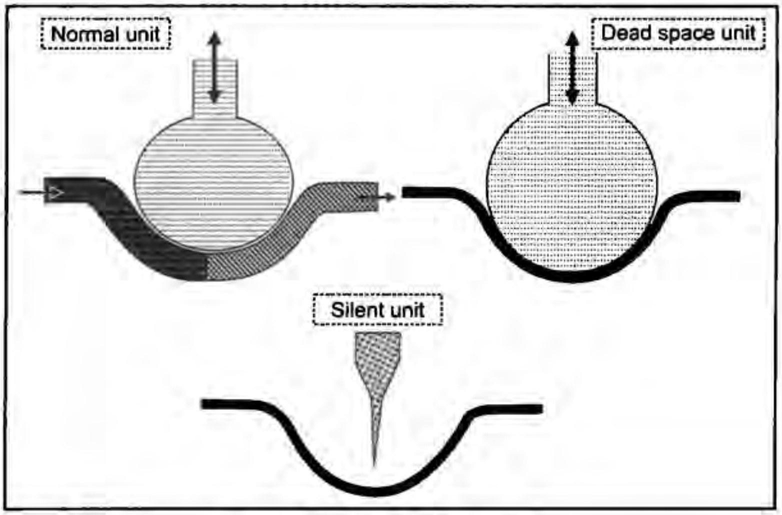


Figure 8.2: Gas exchange unit

- *Normal unit:* In which both ventilation and perfusion are relatively equal.
- *Dead space unit:* In which the alveolus is normally ventilated but there is no blood flow through the capillary.
- *Silent unit:* In which the alveolus has no ventilation and capillary has no perfusion.
- Shunt unit (Fig. 8.3)

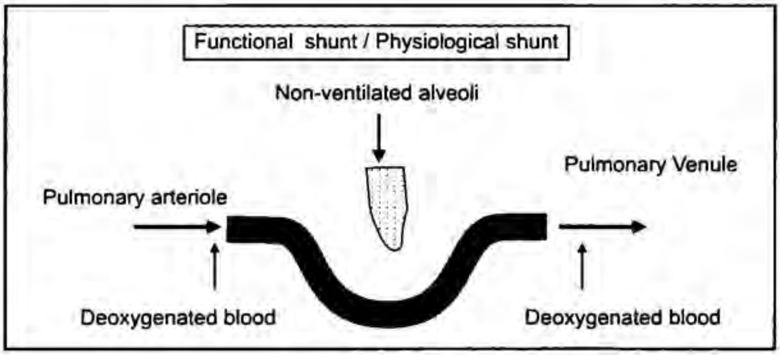


Figure 8.3: Shunt unit

In which the alveolus is completely non-ventilated while the adjacent capillary has a normal blood flow. Blood that supplies poorly ventilated alveoli with low PaO_2 cannot be adequately oxygenated. Therefore, a certain amount of incompletely oxygenated blood enters the arterial circulation and reduces PaO_2 giving rise to physiological shunt.

RELATIONSHIP BETWEEN VENTILATION AND PERFUSION

The Relationship between Ventilation and Perfusion in Normal Lungs and Effects of Gravity

The blood flow is normally greatest in the most dependent parts of the lungs due to the effect of gravity and because the pulmonary circulation is a low-pressure system. Since blood flow is gravity dependent the V_A/Q ratio ranges from about 0.4 in the lower lung zone to 3.0 in the upper lung zone.

The Relationship between Ventilation and Perfusion in Normal Lung (Fig. 8.4)

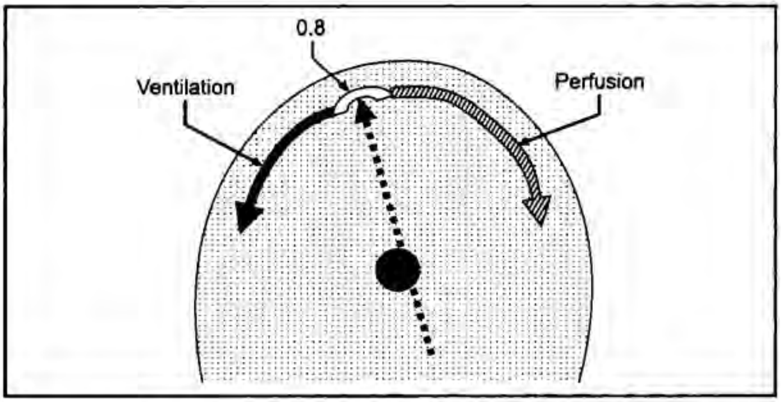


Figure 8.4: Relationship between ventilation and perfusion in normal lung

Normal alveolar ventilation V_A, in an adult, is approximately 4 lit/min and the total perfusion Q (cardiac output) is about 5 lit/min. Therefore, the proportion of ventilation to perfusion is 4/5 = 0.8.

The Relationship between Ventilation and Perfusion in Abnormal Lungs

In Abnormal lungs V/Q ranges from low to high.

- *Low V_A/Q (Fig. 8.5)*

  It indicates shunt perfusion, that is, the alveoli are perfused but not adequately ventilated. Mucous, atelectasis, airway obstruction and interstitial lung disease are examples of those that can cause a decrease in ventilation. If the ratio is low, PaO_2 is markedly reduced and $PaCO_2$ rises a little.

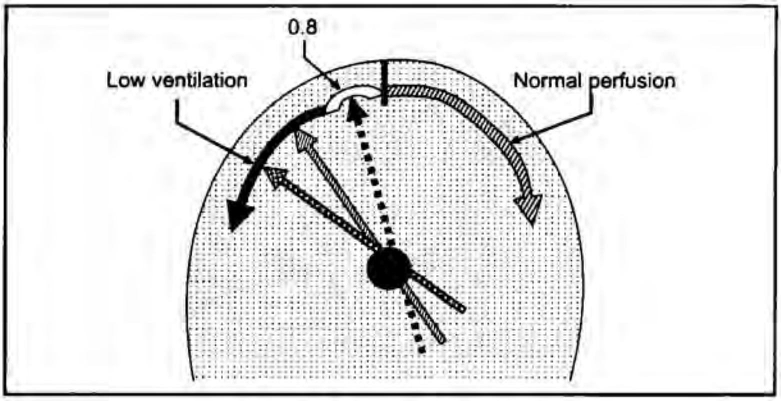


Figure 8.5: Low V/Q –Shunt

- *High V_A/Q (Fig. 8.6)*
  It indicates dead space ventilation, that is, the alveoli are ventilated but not perfused. Pulmonary embolism, pulmonary hyperinflation, hypovolaemia, COPD are examples of those that decrease pulmonary perfusion and lead to high V/Q.

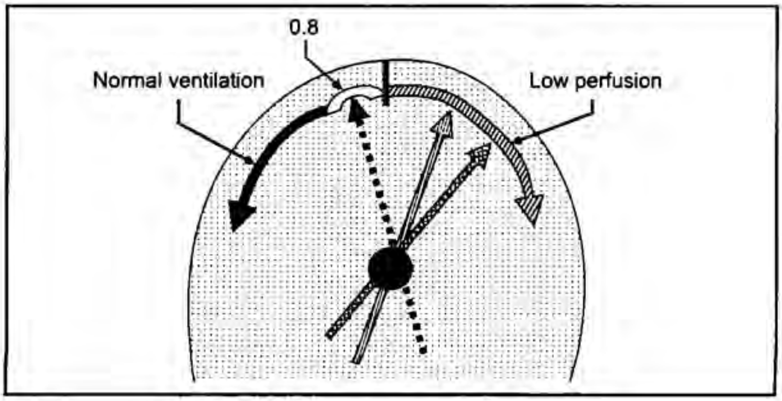


Figure 8.6: High V/Q–Dead space ventilation

ANAESTHETIC APPLICATIONS

1. *Uncomplicated V/Q mismatch*
   V/Q mismatch is responsible for the development of hypoxaemia. With sufficient pulmonary reserve, a patient can usually compensate for the hypoxaemic condition by hyperventilation. Hypoxaemia caused

by uncomplicated V/Q mismatch is readily reversible by oxygen therapy.

2. *Regional differences in the lungs*
   There are normally regional differences in the lungs; in the upright position the ratio in the lung apices is high. A typical value for V/Q in the lung apices is 3 and in the lung bases 0.4. Greater degrees of ventilation perfusion mismatching gives rise to greater than normal variation in the oxygen saturation of the blood in the lungs.
3. *Correction of hypoxaemia caused by V/Q mismatch*
   In mechanical ventilation increasing the rate, tidal volume or F_iO_2 on the ventilator can compensate hypoxaemia caused by V/Q mismatch.

Compensatory Changes in Perfusion for Impaired Ventilation

Impaired ventilation is compensated by a reduction in blood flow to the poorly ventilated alveolus, resulting in better oxygenation of the arterial blood. A low PaO_2 and a raised $PaCO_2$ initiates a blood vessel construction called hypoxic pulmonary vasoconstriction response (HPV response). Due to blood vessel constriction, blood flow to under-ventilated alveoli diminishes. Instead, blood is redirected to well-ventilated alveoli.

No Compensatory Changes in Ventilation for Impaired Perfusion

There is no compensatory mechanism for impaired perfusion. Cardiac output is unevenly distributed within the lungs without a corresponding redistribution in ventilation. Good ventilation of poorly perfused pulmonary perfused section of alveoli continues. This is seen in pulmonary embolus, hypovolaemia and gives rise to a large dead space.

CHAPTER

9

Diffusion Defect

COMPONENTS OF GAS EXCHANGE (FIG. 9.1)

In the normal pulmonary gas exchange, the most important components are:

- Ventilation
- Perfusion
- Diffusion

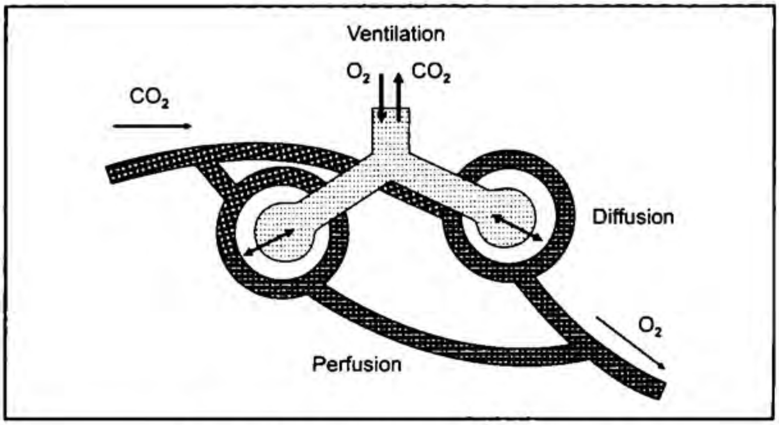


Figure 9.1: Components of gas exchange

Ventilation

Ventilation is the process of gas transport between the alveolus and the atmosphere. Hypoventilation can be due to obstructive, restrictive or other ventilation disorders. Upper abdominal surgery decreases FRC by around 35% reducing all lung volumes giving rise to restrictive ventilation disorder. This gas exchange abnormality is more marked if adequate analgesia is not given.

Perfusion

Severe perfusion disorder can occur due to disorder of pulmonary macrocirculation, producing gas exchange abnormalities, frequently seen in ICU patients. Patients with septicemia, acute pancreatitis and peritonitis develop acute pulmonary failure due to deposit of fibrin clots, granulocyte aggregate in pulmonary microcirculation. Pulmonary embolism, decreased cardiac output due to shock, haemorrhage or cardiac failure are classic examples of perfusion disorder.

Diffusion (Fig. 9.2)

Diffusion can be defined as the delivery of oxygen from the alveoli to the blood and the transfer of carbon dioxide from the blood to the alveoli. Diffusion of oxygen and carbon dioxide across the alveolar capillary (A-C) membrane is due to gas pressure gradients.

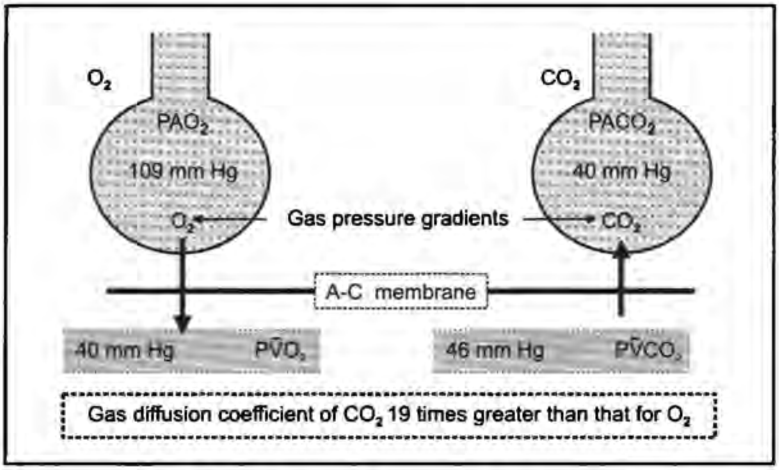


Figure 9.2: Diffusion of oxygen and carbon dioxide

Oxygen Diffusion (Fig. 9.3)

Adequate diffusion of oxygen takes place because of the significant higher partial pressure of oxygen in the air, though oxygen has a relatively poor diffusing capacity. Since the rate of reaction of oxygen with haemoglobin also plays a major role in diffusion, the plasma and the red cell membrane must also be considered in the case of oxygen diffusion.

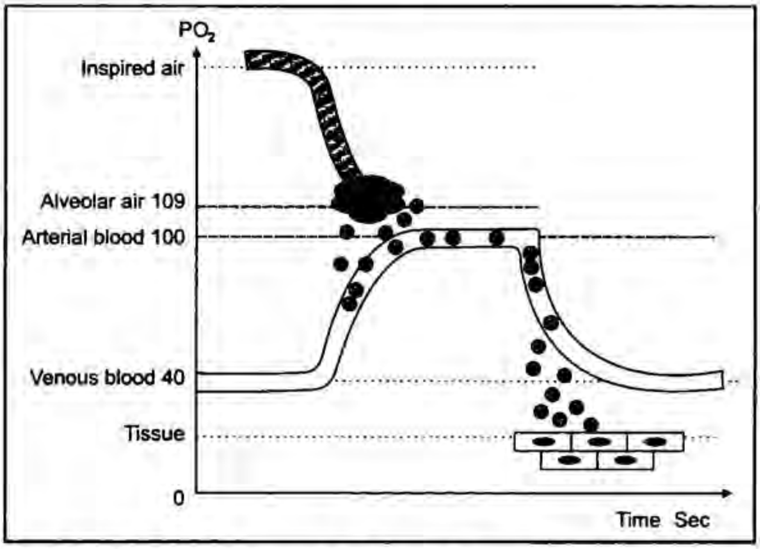


Figure 9.3: Diffusion of oxygen over alveolar membrane and at tissue level (For colour version *see* Plate 2)

Oxygen diffuses from the lungs ($P_AO_2 = 109$ mmHg) to the pulmonary arteries ($P\overline{v}O_2 = 40$ mmHg) with a pressure gradient of 69 mm Hg.

Carbon Dioxide Diffusion (Fig. 9.4)

Though there is hardly any difference in the partial pressure of carbon dioxide in capillary blood and alveolar air, carbon dioxide diffuses in the opposite direction.

Carbon dioxide diffuses from the pulmonary arteries ($P\overline{v}CO_2$= 46 mm Hg) to the lungs ($P_ACO_2 = 40$ mmHg) with a net pressure gradient of

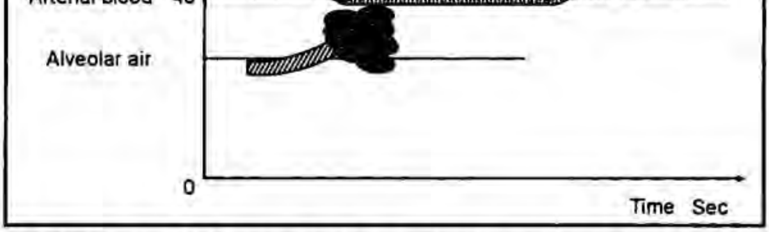


Figure 9.4: Diffusion of Carbon dioxide over alveolar membrane and at tissue level (For colour version *see* Plate 2)

ly 6 mmHg. This is possible because the gas diffusion coefficient for arbon dioxide is 19 times greater than that for oxygen.

A gas exchange disorder can be caused by disturbed diffusion. This sturbance due to a gas exchange failure at the alveolar capillary embrane can take place for a variety of reasons. The rate of transfer s directly proportional to the area of the membrane, the solubility of e gas and the partial pressure gradient across the membrane and versely proportional to the distance across which diffusion has to occur.

FACTORS AFFECTING DIFFUSION OF OXYGEN

Decrease in the Pressure Gradient

Barometric Pressure

Reduced inspired oxygen tension (PIO_2) greatly impairs the diffusion f oxygen. The inspired oxygen tension is directly related to the barometric pressure. At a high altitude the inspired oxygen tension is ow due to low barometric pressure. This leads to a condition known as hypoxic hypoxia.

Smoke Inhalation

Patients who suffer from smoke inhalation are at a risk for developing hypoxic hypoxia. Smoke inhalation gives rise to hypoxia due to the following reasons:

- Consumes oxygen: Combustion consumes oxygen in the air which will reduce PIO_2.
- Impaired lung function: During combustion lung functions are also impaired by the presence of carbon monoxide, toxic gases and inert particles which are found in a fire.

A-C membrane

In addition to the pressure gradient and diffusion coefficient, the gas diffusion rate is also affected by the thickening of the A-C membrane from increased interstitial and alveolar fluid retention e.g.

- Pulmonary oedema
- Noncardiogenic pulmonary oedema -ARDS
- Retained secretions
- Pulmonary fibrosis
- Pneumonia
- Decreased surface area of the A-C membrane, i.e. reduction in gas exchange area
  - Emphysema-destruction of alveoli
  - Atelectasis
  - Embolism
  - Pneumothorax.

Insufficient Time for Diffusion

In patients with fibrosing alveolitis hypoxaemia can occur during exercise. This is due to the inability of the alveolar oxygen to equilibrate with oxygen in the red blood cells in pulmonary capillaries due to rapidity of pulmonary blood flow. Patients having tachycardia will have a short time available for diffusion giving rise to hypoxia.

CLINICAL SIGNIFICANCE OF DIFFUSION DEFECTS

- The above stated conditions induce poor gas diffusion and can severely hinder the oxygenation process.
- The patient will have hypoxaemia and hypoxia. Identification of the mechanism producing hypoxaemia helps to predict the effects of an applied therapeutic measure.
- Severe hypoxaemia and hypoxia may lead to hypoxic pulmonary vasoconstriction, pulmonary hypertension and cor pulmonale.

Hypoxaemia

Hypoxaemia is reduced oxygen in the blood. It is present when the oxygen level (e.g. PaO_2, SaO_2) is decreased in arterial blood.

- Though PaO_2 is most often used to evaluate a patient's oxygenation status, it only reflects the oxygen that is dissolved in the plasma; it does not represent all the oxygen carried by the blood, i.e. by haemoglobin and plasma.
- For precise assessment oxygen content should be used because it includes the oxygen combined with haemoglobin as well as oxygen dissolved in the plasma.

Hypoxia

Hypoxia is reduced oxygen the body organs and tissues. This is due to the failure of tissues to receive adequate quantities of oxygen. Hypoxaemia reflects the likelihood of hypoxia, but it is important to remember that hypoxia can occur in the absence of hypoxaemia with normal PaO_2.

- Cyanide poisoning causes histotoxic hypoxia in which the tissues cannot utilize oxygen.
- In anaemic hypoxia, the low haemoglobin level causes low oxygen content while the PaO_2 may be normal.

Since PaO_2 measures the oxygen tension of the plasma only, it cannot be used for assessment of anaemic or histotoxic hypoxia. Arterial oxygen content (CaO_2) should be measured and used to assess the oxygenation status of a patient. In addition to the PaO_2 and CaO_2 measurements, hypoxia produces some clinical signs that can be used as a secondary assessment tool, though the presence of hypoxia (decrease PaO_2 in organs and tissues) may not be always apparent.

Signs of Hypoxia

- In most clinical situations hypoxaemia is readily corrected by a moderate amount of supplemental O_2.
- The important clinical signs of oxygenation failure and hypoxia include hypoxaemia, dyspnoea, tachypnoea, tachycardia and cyanosis.

OXYGENATION FAILURE

Oxygenation failure is defined as severe hypoxaemia (PaO_2 < 40 mm Hg) that does not respond to moderate to high levels (50-100%) of supplemental O_2.

Causes of Oxygenation Failure

- Hypoventilation
- V/Q mismatch
- Intrapulmonary shunting

Regardless of the etiology of oxygenation failure, mechanical ventilation may be needed to minimize the work of breathing and provide oxygenation support.

CHAPTER

10

Lung Volumes and Capacities

LUNG VOLUMES (FIG. 10.1)

1. *Tidal volume (V_T):* It tidal volume is the amount of air passing in and out of the lungs and respiratory passages during quiet breathing. That is why volume varies with the size of the airway, which depends on age and the depth of respiration. It is around 19 ml in the average newborn infant and 450-750 ml in resting adults. The normal tidal volume is 6-8 ml/kg regardless of age.
2. *Inspiratory reserve volume (IRV):* The maximum volume of air that can be inspired after normal inspiration. IRV is the difference between normal and maximal inspiration. IRV is about 2.5 lit or 2/3rd of the vital capacity.
3. *Expiratory reserve volume (ERV):* The maximum volume of air that can be expired after normal expiration. ERV is about 1.5 lit or 1/3rd of the vital capacity.
4. *Residual volume (RV):* It is the volume of air present in the lungs at the end of maximum expiration. This air cannot be expelled from the lungs even after maximum effort. Normal value of RV is about 1.5-2 lit.

Static Lung Volumes

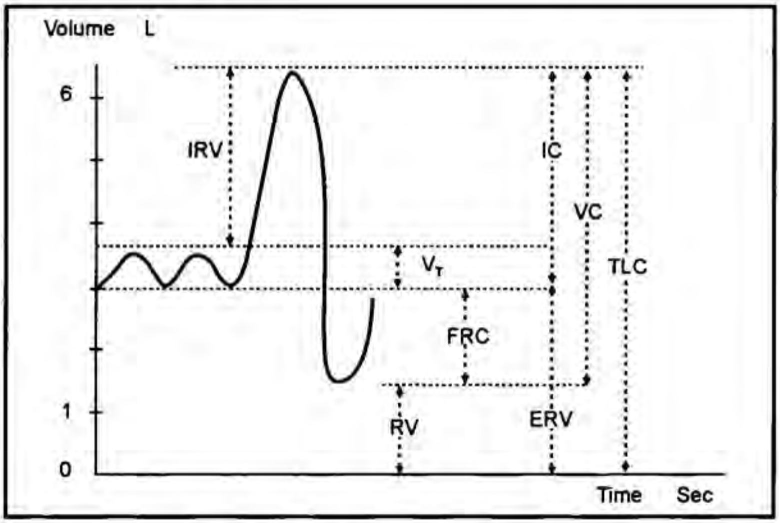


Figure 10.1: Static lung volumes

LUNG CAPACITY (FIGS 10.2 AND 10.3)

1. *Vital capacity-VC*

   The maximum volume of air that can be expired after maximum inspiration. It is therefore a measure for the largest possible breath excursion.

   The normal vital capacity is about 30-40 ml/kg in infants and 45 to 55 ml/kg (3.5-4.5 lit) in adults.

2. *Inspiratory capacity* $IC - IRV + V_T$

   The maximum volume of air that can be inspired after normal expiration.

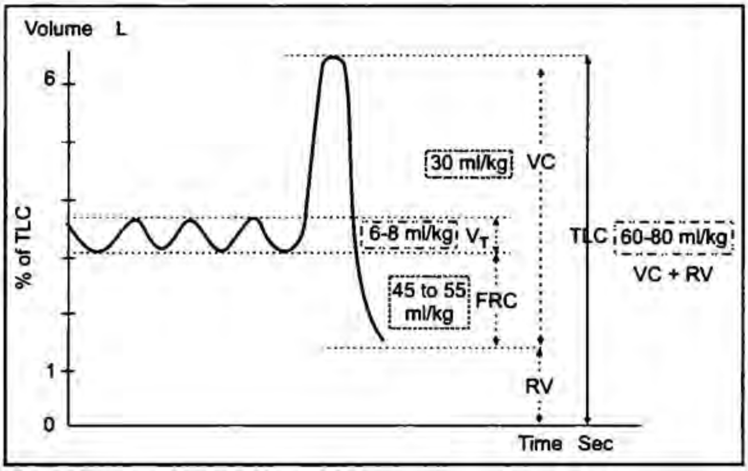
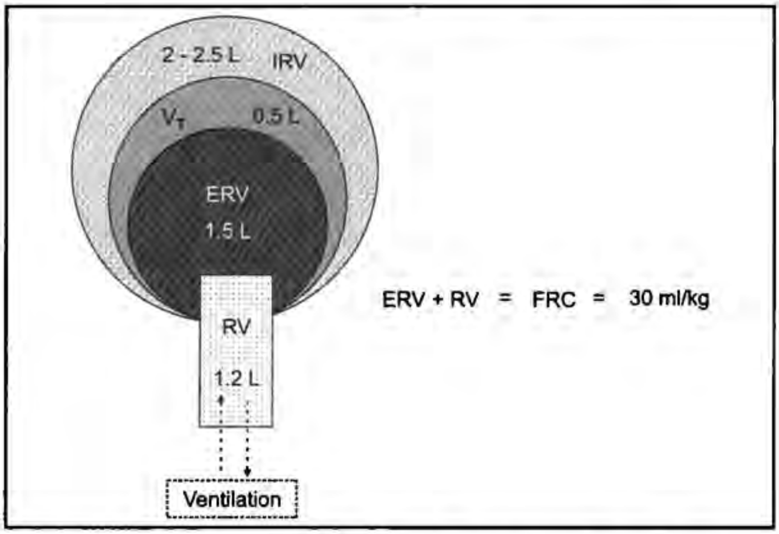


Figure 10.2



Figures 10.2 and 10.3: Static lung volumes and capacities
(For colour version *see* Plate 3)

3. *Total lung capacity-TLC*
   Total lung capacity or TLC is the volume of gas present in the lungs after maximal inspiration. The normal range for total lung capacity is 60-80 ml/kg (approximately 6 lit). It can be calculated from VC + RV.
4. *Functional residual capacity (FRC) (Figs 10.4 and 10.5)*

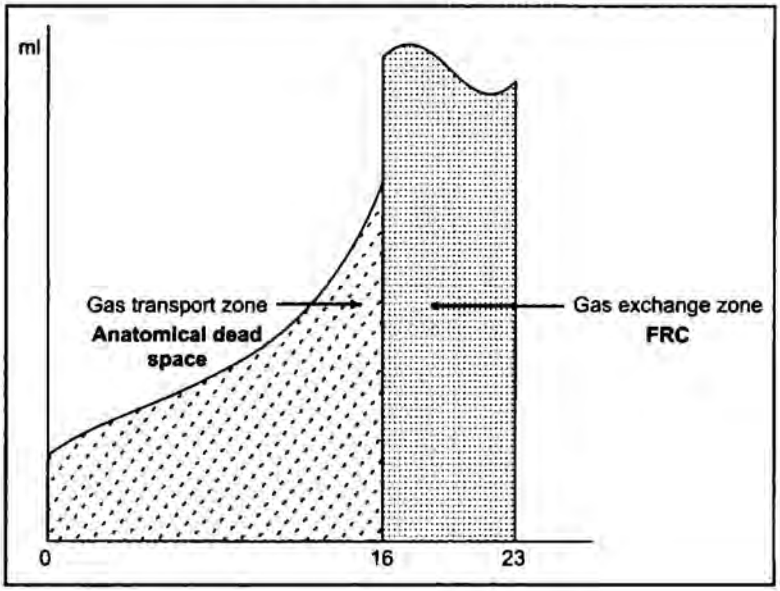


Figure 10.4: Gas exchange zone –FRC

FRC is defined as the volume of gas in the lungs that exists at the end of a normal expiration when there is no airflow and alveolar pressure equals the ambient pressure. It can be considered as a measure of gas exchange area. The normal FRC is about 30 ml/kg (3 to 3.5 lit). Functional residual capacity is the sum of ERV + RV. FRC results from the balance between the forces that favour alveolar collapse and those that maintain alveolar inflation.

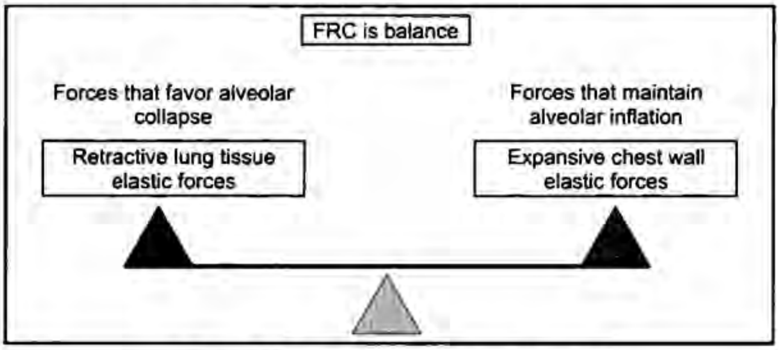

FRC is balance
Forces that favor alveolar collapse
Retractive lung tissue elastic forces
Forces that maintain alveolar inflation
Expansive chest wall elastic forces

Figure 10.5: Functional residual capacity

Under these conditions retractive lung tissue elastic forces exactly balances expansive chest wall elastic forces.

Pathophysiology of Acute Restriction of All Lung Volumes after Surgery

All lung volumes decrease significantly during and after surgery, performed under general anaesthesia, especially after upper abdominal surgery and thoracotomy.

Mechanism of Decrease of FRC during Anaesthesia

The FRC falls by 20 % within a few minutes after initiation of anaesthesia and 30-35% postoperatively. This decrease is probably caused by:

- In the supine position, there is a elevation of the diaphragm which increases intra-abdominal pressure
- Loss of tone of respiratory musculature
- An increase in the central blood volume.

Mechanism of Decrease of FRC Postoperatively

- If an adequate analgesia is not given, especially for upper abdominal surgery, associated pain will give rise to shallow breathing
- Cough suppression
- Surgery induced dependent atelectasis

- Increased intra-abdominal pressure due to various causes
- Residual effects of narcotics and muscle relaxants.

Degree of Restriction

The degree of restriction is characterized by:

- Reduction of the inspiratory reserve volume to about 10% of the original volume
- Reduced vital capacity to about 50-75%
- Reduction of FRC by about 30-35%

Reduction of FRC leads to a smaller closing capacity FRC < CC. There is fall of FRC below critical closing volume giving rise to small airway closure occurring at the end of expiration. Intermittent closure of bronchioles leads to an increase in intrapulmonary right to left shunt, reducing oxygenation. Postoperative lung function returns to normal only after 2-3 weeks.

Clinical Significance of Normal Vital Capacity and Effective Cough

When planning perioperative management of anaesthesia one should remember that over 30% of patients develop postoperative respiratory failure if the perioperative vital capacity is less than 50% of the normal value or less than 1.75-2 lit in adults.

The patient breathes superficially and may not be able to cough effectively if the vital capacity is not three times the tidal volume (normal value 8 ml/kg). Patient will have retention of secretions with consequent development of atelectasis and secondary pneumonia due to ineffective coughing.

Precautionary Measures to Avoid Complications

- Adequate postoperative analgesia
- Preoperative respiratory therapy

In view of above complications it is necessary to keep the FRC above closing volume so that the sufficient inspiratory and expiratory gas exchange area is maintained.

Clinical Significance of Decreased FRC

1. Development of dependent atelectasis
2. Small airway closure
   - During anaesthesia a reduction in resting lung volume takes place, leading to closure of some smaller airways during inspiration and expiration.

- Airway closure can be inhibited by increasing lung volumes, for example by ventilation with PEEP.

3. Reduced ventilation – perfusion ratio which results from:
   - Reduced compliance
   - Increased resistance
   - Increased (A-a)O_2 resulting from the perfusion of atelectatic compartments
   - The reflex mechanism due to hypoxic pulmonary construction which redistributes the flow of blood from unventilated to ventilated areas is often depressed during anesthesias.

FRC and Newborn (Fig. 10.6)

The chest cage offers little opposition to collapse at end-expiration; this explains why the newborn, and especially the premature infant, have low FRC and Thoracic Gas Volume (TGV).

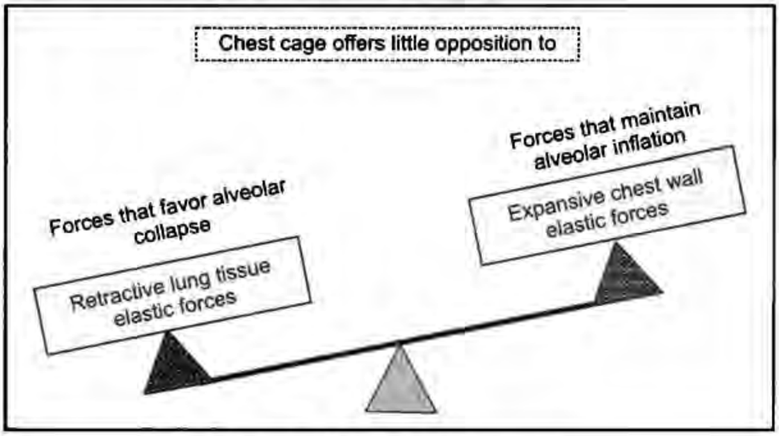

Chest cage offers little opposition to
Forces that favor alveolar collapse
Retractive lung tissue elastic forces
Forces that maintain alveolar inflation
Expansive chest wall elastic forces

Figure 10.6: FRC and newborn

CLOSING VOLUME (CV)

In a fully inflated lung all the airways are open. The lung volume at which smaller airways start closing during expiration is known as the closing volume (Fig. 10.7).

Closing Volume and Newborn

In newborns the closing volume may occur above FRC. In conjunction with the underdeveloped walls of the conducting airways it may explain the tendency for early airway closure and gas trapping in premature infants.

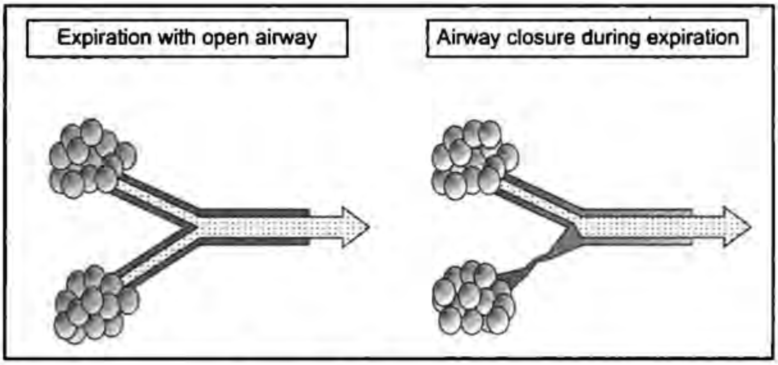


Figure 10.7: Closing volume

CLOSING CAPACITY

CC = CV + RV

Closing capacity is the sum of the closing volume and the residual volume. It is advisable that CC to be expressed as a percent of the total lung capacity.

AIRWAY CLOSURE

Factors Influencing and Increasing Closing Volume/Airway Closure

1. *Pre-existing Disease*

   Chronic obstructive pulmonary disease.

2. *Age (Fig. 10.8)*

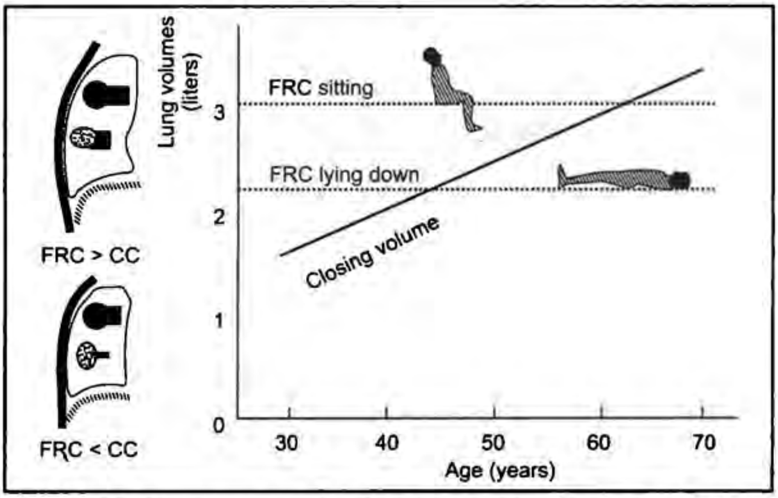


Figure 10.8: Closing volume and FRC in relation to age and body position (For colour version *see* Plate 3)

- *Newborn:* In the newborn airway closure occurs within the lung volumes of normal breathing.
- *Children:* In children older than 6 years FRC exceeds CC. However, in infants and in children less than 6 years, CC exceeds FRC. This explains the tendency for atelectasis in young children.
- *Adults:* In normal, young, healthy individuals one can expect all airways to be open during normal breathing. In healthy individuals at a mean age of 44 years CC = FRC in the supine position and at a mean age of 66 years CC = FRC in the upright position.

3. *Position (Fig. 10.8)*

- Airway closure occurs primarily in the more dependent parts of the lungs.
- Closure of the bronchioles occurs, especially in the dependent (dorso – basal) pulmonary segments, where the extraluminal tissue pressure exceeds the endobronchial airway pressure due to gravity.

4. *Relationship between FRC, CV and CC*

When FRC exceeds CC, the small airways and the alveoli remain open and airways do not close at midexpiratory pressure. On the other hand, when CC exceeds FRC the small airways and alveoli tend to collapse during tidal ventilation.

When CC>FRC, at the end of normal expiration, closure of the small airways and alveoli will occur giving rise to an increased shunt in correlation with the breathing cycle and absorption atelectasis develop.

The relationship between FRC and CC is far more important than consideration for FRC or CC alone because that determines whether a given respiratory unit is normal or atelectatic or has a low V/Q ratio.

Relationship between FRC and CC (Fig. 10.9)

The relationship between the FRC (which is the % of total lung capacity that exists at the end of the exhalation), shown by the level of each trough of the sine wave tidal volume and the CC of the lung (three different closing capacities are indicated by the three different, straight lines).

Anything that decreases FRC relative to CC or increases CC relative to FRC will convert normal areas to low V/Q and atelectatic areas. Development of low V/Q and atelectatic areas will cause hypoxaemia.

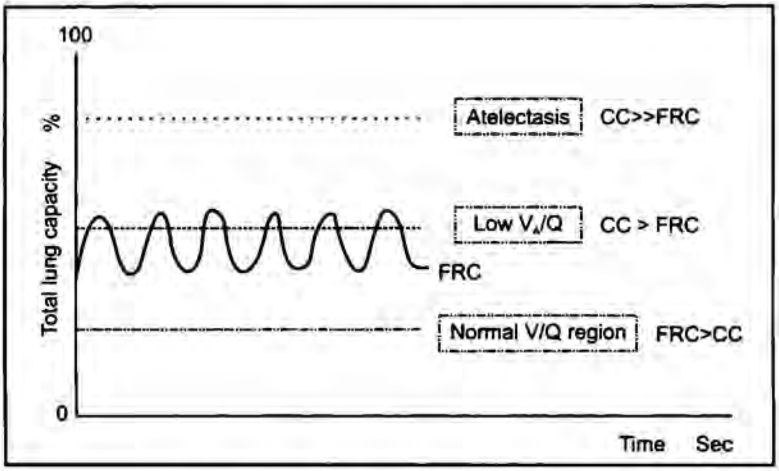


Figure 10.9: Relationship between FRC and CC

- *Normal V/Q region:* If the closing volume of the lungs is below the whole of tidal respiration, then no airways are closed at any time during tidal respiration. This is under normal circumstances.

- *Low V/Q region:* If the closing volume of some airways lies within the tidal volume, then as lung volume increases during inspiration, some previously closed airways will open for a short period of time until lung volume once again recedes below the closing volume of these airways.

  Due to these opening and closing, airways are open for a shorter period of time than normal airways; they have less chance or time to participate in fresh gas exchange, which is equivalent to a low V/Q region. In such a patient oxygenation of blood can be maintained by giving a high-inspired oxygen concentration.

- *Atelectasis:* When the volume of lung at which some airways close is greater than the whole of the tidal volume, then lung volume will never increase enough during tidal inspiration to open any of these airways. Thus, these airways stay closed during all of the tidal respiration. Airways that are closed all the time are equivalent to atelectasis leading to shunt phenomenon. This requires a different strategy other than increasing inspired oxygen concentration.

NORMAL RESPIRATORY VALUES

A similarity in respiratory function between neonates and adults exists in different parameters relative to body weight-as in tidal volume V_T, dead space-tidal volume ratio V_T/ V_D and FRC.

		Newborn	*Adult*
Respiratory frequency breaths/min		30-40	12-16
Tidal volume ml/kg	V_T	6-8	7
Dead space ml/kg	V_D	2–2.5	2.2
Dead space/Tidal volume ratio	V_T/V_D	0.3	0.3
Vital capacity ml/kg	VC	35-40	50-60
Functional residual capacity ml/kg	FRC	27-30	30
Total lung capacity ml/kg	TLC		60-80
Lung compliance ml/cm/H_2O	C_L	5-6	200
Alveolar ventilation ml/kg/min	V_A	100-150 (newborn's higher metabolic rate)	60
Oxygen consumption ml/kg/min	VO_2	6-8	3
Airway resistance cm/H_2O/l/sec	R_{AW}	25-30	1.6
Closing Capacity		CC>FRC (less than 6 yrs)	FRC > CC (more than 6 yrs)
Tracheal diameter mm		3.6-4	13-23

CHAPTER

11

Work of Breathing

PRESSURE, FORCE AND WORK OF BREATHING (Fig. 11.1)

Pressure (P) is physically defined as the force (F) exerted on an area (A) Pressure is expressed in cmH_2O/mbar

$$\text{Pressure} = \frac{\text{Force}}{\text{Area}}$$

The force can thus be calculated by the formula:

$$\text{Force} = \text{Pressure} \times \text{Area}$$

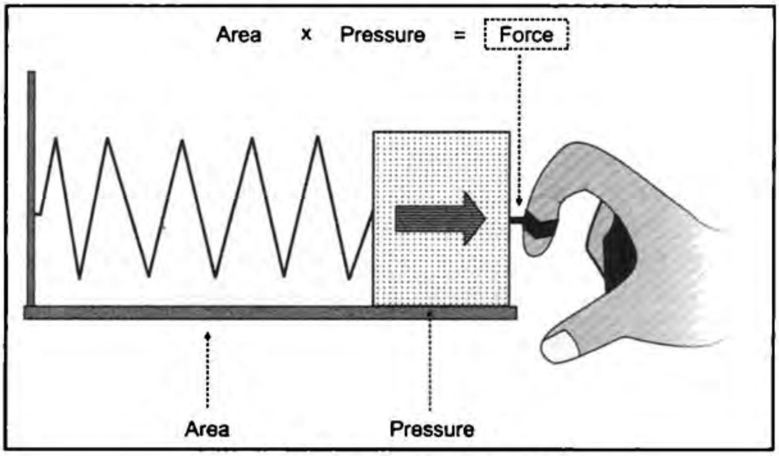


Figure 11.1: Force = Pressure x Area

Work is said to be done when a force causes an object on which it is acting, to move and is physically defined as force multiplied by distance.

Work = Force × Distance

Work of Breathing (WOB) = Pressure × Area × Distance

Area multiplied by *distance* results in a dimension of *volume*, so that:

WOB = Pressure x Volume

Resistance and Friction

The horizontal force moving the block in the direction of the arrow is opposed by two factors:

- The elastic resistance of the spring (Fig. 11.2).
- The friction between the block and the surface upon which it is moved (Fig. 11.3).

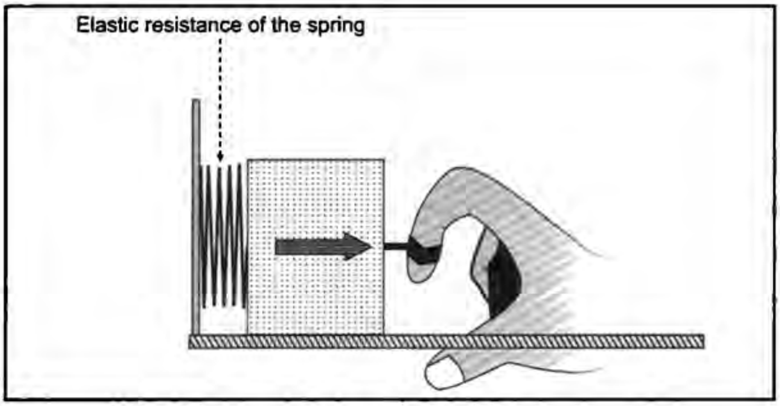
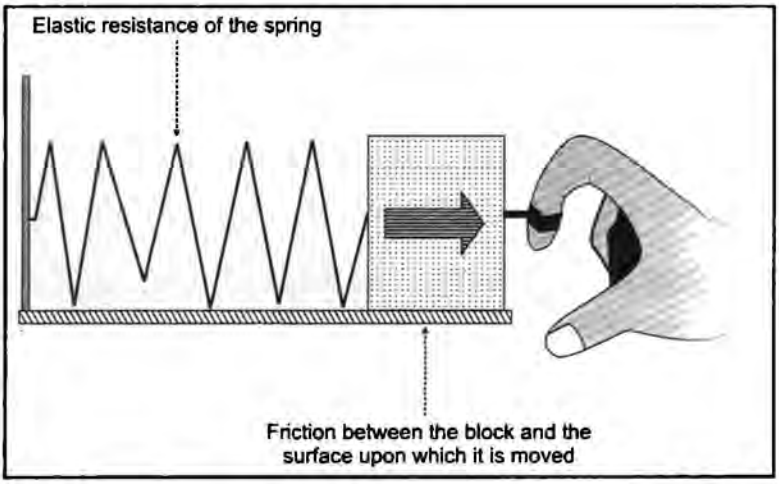


Figure 11.2


Elastic resistance of the spring
Friction between the block and the surface upon which it is moved

Figures 11.2 and 11.3: Opposition of force for moving the block

Force Required to Overcome the Spring and the Friction of the Surface (Fig. 11.4)

- The force required to overcome the spring
  This relates to the force required to overcome the elastic resistance of the spring to the distance. This component follows the behavior of elastic structures i.e. force is directly proportional to the distance moved. Hence, the pressure volume curve is a straight line if not for the second component of the work of breathing namely work required for viscous resistance.

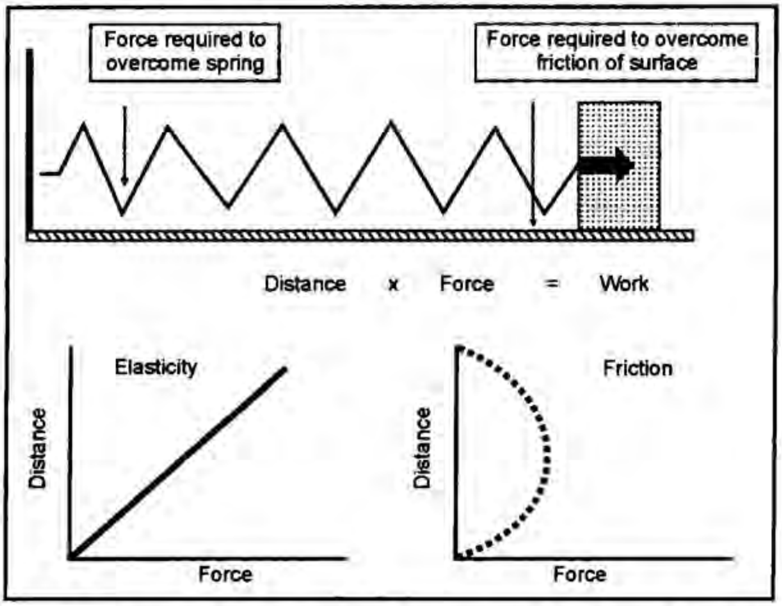

Force required to overcome spring
Force required to overcome friction of surface
Distance x Force = Work
Elasticity
Distance
Force
Friction
Distance
Force

Figure 11.4: Force required to overcome the spring and the friction of the surface

- Force required to overcome the friction of the surface
  This represents the force required to overcome the friction of the surface on which the block is moved. Since work = force x distance, the area enclosed between the line representing force and the coordinate (representing distance), represents the work performed.

COMPONENT OF WORK OF BREATHING (Fig. 11.5)

The total work of breathing has two broad components.

The crosshatched area to the left of the diagonal represents the work done to overcome the elastic resistance of the spring. The stippled area to the right of the diagonal represents the work done to overcome the friction.

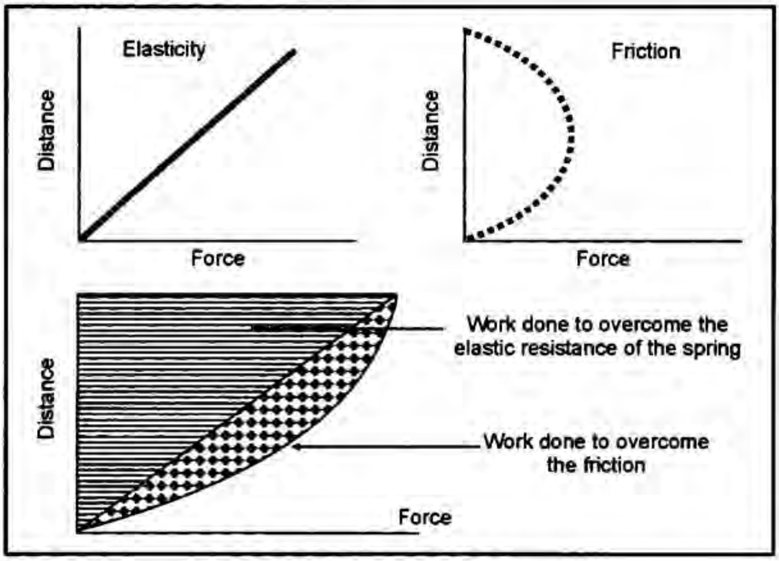

Elasticity
Distance
Force
Friction
Distance
Force
Distance
Work done to overcome the elastic resistance of the spring
Work done to overcome the friction
Force

Figure 11.5: Total work required to move the block (For colour version *see* Plate 4)

The sum of these two areas represents the total work that must be performed to move the block through the given distance.

1. *A spring of greater tension means an increase in the elastic workload (Fig. 11.6):*
   If our spring were now to be substituted with a spring of greater tension, a greater force would be required (and more work would have to be performed) to move the same block the same distance over the same surface.

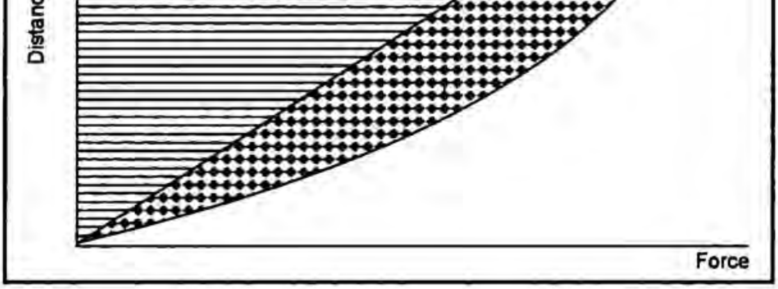


Figure 11.6: Increase in elastic workload

This greater force is depicted by the pivoting downward (towards the force axis) of the diagonal representing force. Though the area representing the work done in overcoming friction does not change, there is an increase in the area representing the work done to overcome the elastic resistance of the spring, which increases the area of the total work done.

2. *An increase in the friction workload (Fig. 11.7):*

   Similarly, if friction alone were to be increased while leaving the tension of the spring unchanged, the stippled area would increase correspondingly and though the work done to overcome the elastic resistance of the spring does not change, the total work increases.

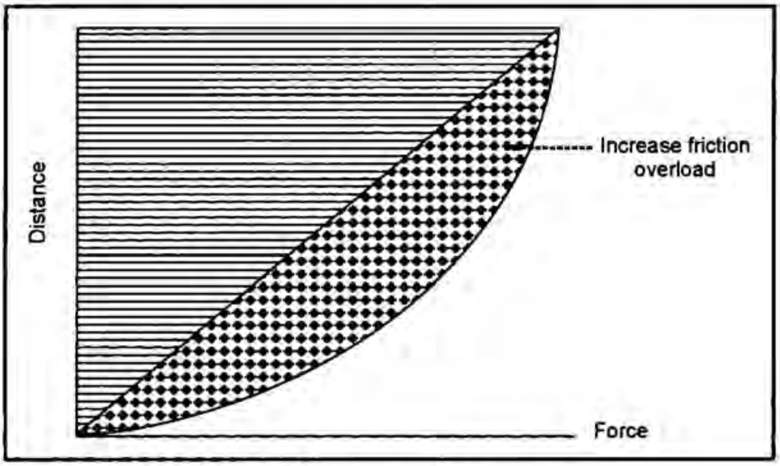


Figure 11.7: Increase friction overload

FACTORS RESISTING LUNG EXPANSION

As in the example of the block and the spring, there are two components (elastic and nonelastic) that resist lung expansion and pressure needs to overcome these components before the lung volume can increase. WOB refers to the work generated by the patient or the ventilator to overcome the elastic and resistive properties of the respiratory system which is normally performed during inspiration.

Elastic Work

The elastic recoil of the lung is defined as the force tending to return the lung to its original size after stretching. One component of WOB is the elastic work done to overcome elastic recoil of the lung and the chest wall. It is not the force required in expanding the lung, as this is also a measure of the rigidity of the lung, e.g. pulmonary congestion. Stiffer the lungs more the elastic work to be performed.

Non-elastic (Viscous) Work

The viscous resistance is a frictional force, and therefore depends on the velocity of motion. That is why the work done to overcome viscous resistance is maximum at the middle of inspiration when velocity of airflow is the highest. Also, this component of the work of breathing is increased if the velocity of airflow increases, as during hyperventilation. The work done to overcome viscous resistance can be further split into two components:

Airway Resistance

Most of the viscous or frictional resistance is attributable to the airway resistance. That is why the work done to overcome viscous resistance increases as in obstructive lung diseases. The airway resistance is important because it is dependent on the length and size of the lumen of the bronchial tree. The anaesthetist adds an extra airway resistance when he inserts an endotracheal tube. This is more important and significant in the intubation of a young child, because the "narrower" the airways more is the friction.

Tissue Resistance or Structural Resistance

A small part of frictional resistance is made up by tissue resistance i.e. friction between layers of tissues, which is composed of the thoracic wall, the diaphragm and the abdominal contents. It is related to the speed of the flow of air so that when this is maximal so is the structural resistance.

Work = Force × Distance

WOB = Pressure × *Area* × *Distance*

WOB = Pressure × *Volume*

= Change in pressure × Change in volume

Work (WOB per unit time) = P cm H_2O × V (minute volume in ml)

P-is the total pressure change during the respiratory cycle in cm H_2O

V-is minute volume in ml.

In the process of breathing, force is analogous to the change in pressure (transrespiratory pressure) and distance corresponds to change in volume (tidal volume).

PRESSURE VOLUME CURVE OF THE LUNG (FIG. 11.8)

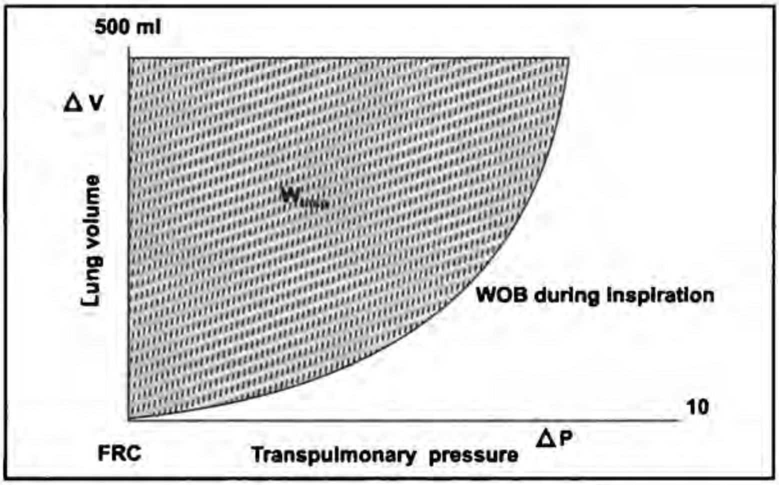


Figure 11.8: Pressure volume curve of the lung

Components of Work of Breathing during Inspiration (Fig. 11.9)

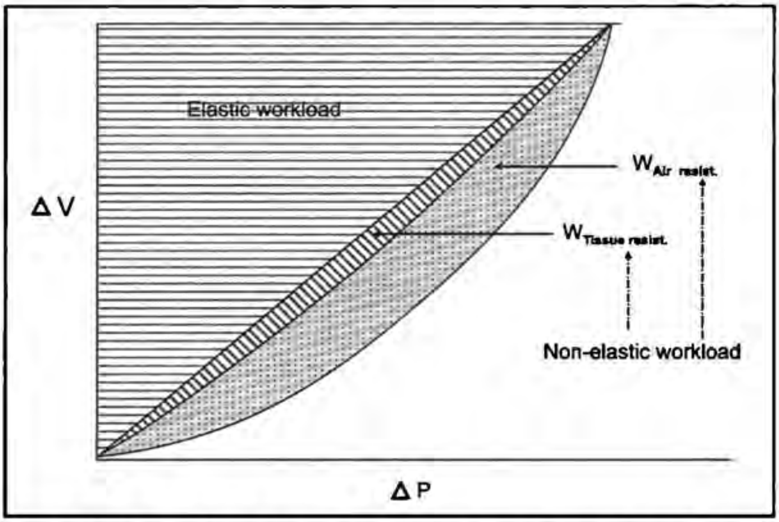


Figure 11.9: Work done during inspiration (For colour version *see* Plate 4)

The horizontal lines represents the elastic workload and the vertical dotted line the non-elastic workload.

Work Done during Expiration (Fig. 11.10)

The solid line with the arrowhead is the inhalation loop while the interrupted line is the exhalation loop. No work is performed during a normal exhalation which is why that loop is "buried" in the area of inspiratory workload.

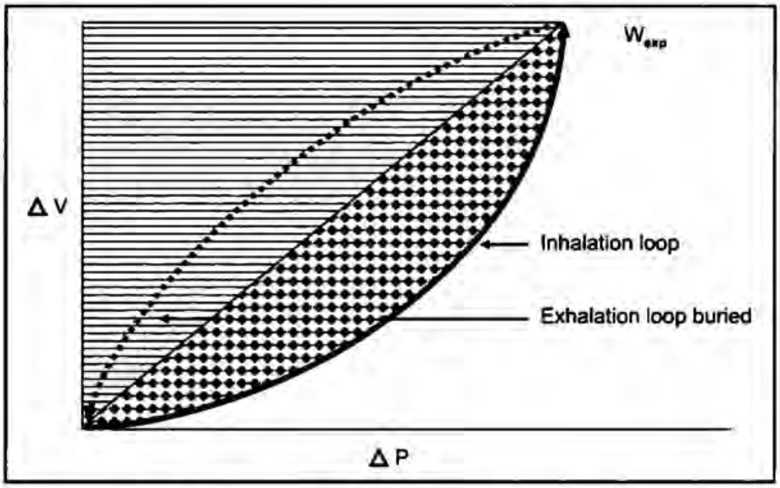


Figure 11.10: Work done during expiration

MEASUREMENT OF WOB

The work of breathing is expressed in Joules. Normal value of WOB for normal resting individuals is 2.5-3 J/min. 10-15 J/min is considered to be the maximum sustainable work of breathing, above this level patients require respiratory support.

WOB comprises all the energy required to ventilate the lungs. The lung is a hollow, distensible structure like a balloon, which is inflated by the application of a positive transrespiratory pressure. During normal tidal breathing, the work of breathing is performed entirely by the inspiratory muscles and almost all of the work is performed during inspiration.

The respiratory muscles do not perform any work during expiration because it is done by the elastic recoil of the lungs and thoracic cage. In fact the energy of elastic recoil is capable of doing much more work than is required to overcome viscous resistance. But under pathological conditions (e.g. expiratory obstruction, forced expiration or in CPAP/PEEP) respiratory muscles have to do the work.

Transpulmonary/Transrespiratory Pressure

The pressure-volume characteristics of the lung also determine the work of breathing, because Work = Pressure x Volume.

Muscle pressure is transpulmonary pressure [airway pressure minus body surface pressure (atmospheric pressure)], generatedby the ventilatory muscles to expand the thoracic cage and lungs. Transpulmonary pressure is pressure needed to drive gas to the alveolar level and also to distend the lung and chest wall. The difference between airway pressure (atmospheric pressure in spontaneous ventilation) and pleural pressure is called transpleural pressure, which determines tidal volume. In other words transpulmonary pressure is also equal to change in pleural pressure. When alveolar pressure exceeds intrapleural pressure, transpulmonary pressure is positive.

Spontaneously Breathing—During Inspiration

In the normal, spontaneously breathing adult individual, during inspiration, alveolar pressure is slightly negative (i.e. subatmospheric) while intrapleural pressure is more negative. Transpulmonary pressure is therefore positive (less negative is more positive than more negative!). This causes the lung to be inflated. During normal inspiration, transpulmonary pressure increases from 0-5 cm H_2O while 500 ml of air is drawn into the lungs.

Intermittent Positive Pressure Ventilation—During Inspiration

In intermittent positive pressure ventilation, positive (i.e. supra-atmospheric) pressure is applied to the alveoli through the airway, creating a positive transpulmonary pressure during inspiration.

WOB AND AIRWAY RESISTANCE OR COMPLIANCE (Fig. 11.11)

Increased airway resistance and decreased chest wall or lung compliance increase the transpulmonary pressure (Ptp) required to inflate the lungs.

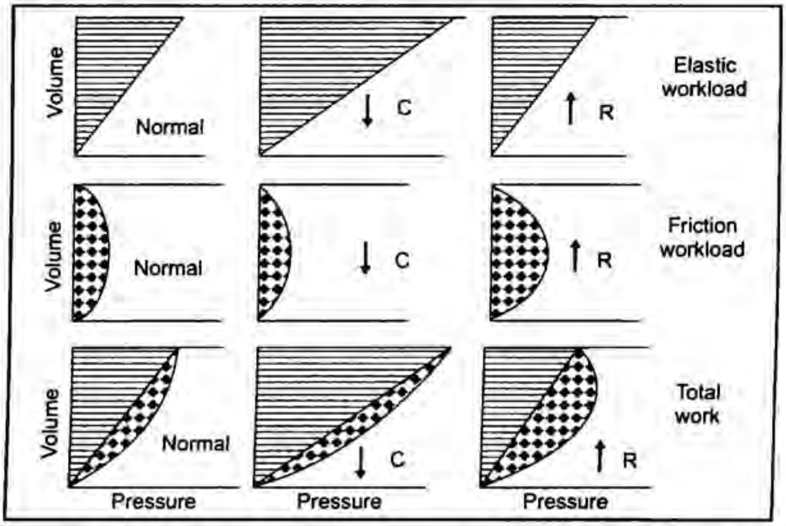


Figure 11.11: The effects of decreasing lung compliance and increasing airway resistance on WOB (For colour version *see* Plate 5)

This imposes a greater workload on the respiratory muscles and increases the oxygen cost of breathing. When the oxygen supply-demand balance of the respiratory muscles is disturbed, respiratory failure may occur because of muscle fatigue. Unfortunately, the task of the respiratory muscle is not simply the inflation of a passive balloon i.e. the lungs, but also to overcome forces which tend to prevent the lungs from being inflated.

Energy Cost of Breathing

Under normal conditions the O_2 consumption for the work of breathing is about 5 lit/min.

During quiet breathing, a healthy person spends less than 5% of his total oxygen consumption on breathing. This fraction can increase enormously and may be the limiting factor for the breathing effort during exercise or in case of lung disease.

During deep and increased breathing, the respiratory work can be increased even up to 20% of total oxygen consumption. In a patient with COPD, oxygen consumption can be so high that the oxygen for the rest of the body becomes compromised. If the energy demand becomes more than the energy supply, it gives rise to metabolic deficit leading to respiratory muscle fatigue. Once muscles are fatigued they can no longer pump hard to maintain sufficient alveolar ventilation. This leads to respiratory pump failure leading to pulmonary insufficiency with hypercapnia.

Types of Lung Diseases

Restrictive Lung Diseases

In restrictive lung diseases, i.e. diseases in which distensibility or compliance) of the lungs, may be reduced, e.g. fibrosis of the lungs, the elastic components of the work of breathing is increased.

Obstructive Lung Disease

In obstructive lung disease, i.e. diseases in which airway resistance may be increased, e.g. bronchial asthma, the work required to overcome viscous resistance is increased.

MAGNITUDE OF THE COMPONENT OF THE WOB (FIG. 11.12)

Magnitude of the Elastic Component of the WOB

The magnitude of the elastic component of the WOB depends on the degree of expansion of the lungs. Patients with restrictive lung disease such as pulmonary fibrosis, pulmonary oedema, and pulmonary compliance decreases leading to high elastic resistance. In this patient

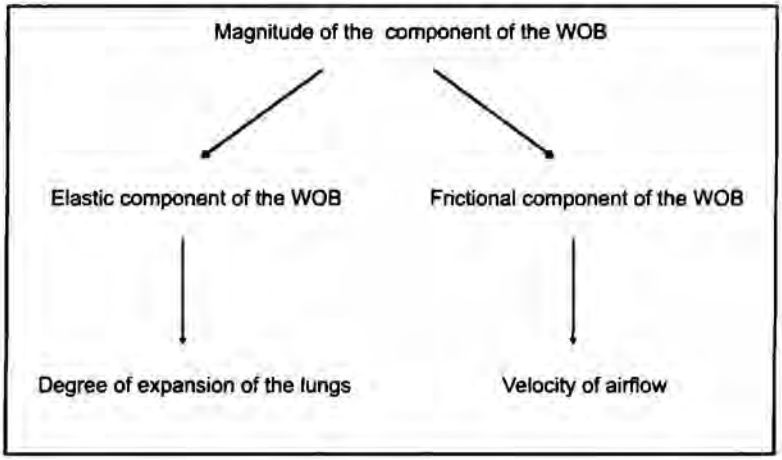

Magnitude of the component of the WOB
Elastic component of the WOB
Frictional component of the WOB
Degree of expansion of the lungs
Velocity of airflow

Figure 11.12: Magnitude of the component of the WOB

the work performed is increased to overcome increased elastic resistance. Therefore, patients with restrictive lung diseases tend to take shallow breaths. In order to achieve satisfactory alveolar ventilation, they increase the frequency of breathing. Thus, the most economical (in terms of energy cost) and therefore the most convenient (i.e. least tiring) method of breathing for patients with restrictive lung disease is rapid and shallow breathing. Expiration is normally passive, but in patients with lung diseases it may become active. The expiratory work must then be included in total WOB.

Magnitude of the Frictional (Viscous) Component of the WOB

In a patient with obstructive airway disease, the magnitude of the frictional (viscous) component of the work of breathing depends on the velocity of airflow. In this patient the work performed is increased to overcome increased airway resistance. Therefore, patients with obstructive lung diseases tend to take slow breaths. When airway resistance is high (asthma, obstructive lung disease), the optimum frequency is decreased and slow deep breaths are favoured. They achieve satisfactory alveolar ventilation by increasing the tidal volume. Hence, the most efficient and convenient method of breathing for patients with obstructive lung diseases is slow and deep breathing.

Patients with different types of lung diseases adopt a pattern of breathing that is most economical for them in terms of oxygen cost of breathing. If a constant volume is maintained:

- Compliance work is increased when the breathing is slow and deep
- Resistance work is increased during rapid shallow breathing.

The below diagram shows the work done against elastic and airflow resistance separately and are summated to indicate the total work of breathing at different respiratory frequencies.

Magnitude of Normal Elastic and Resistive Component (Fig. 11.13)

The total work of breathing has a minimum value at about 15 breaths per minute under normal circumstances.

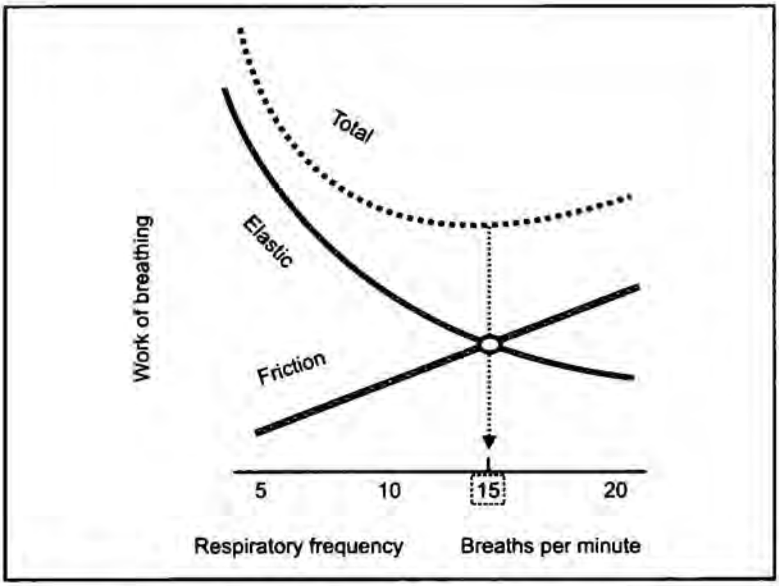


Figure 11.13: Normal elastic and resistive component

Magnitude of Increased Elastic Resistance (Fig. 11.14)

For the same minute volume minimum work is performed at higher frequencies with stiff (less compliant) lungs and at lower frequencies when the airflow resistance is increased.

Magnitude of Increased Obstructive Resistance (Fig. 11.15)

For the same minute volume minimum work is performed at lower frequencies when the airflow resistance is increased.

COMPARISON OF NORMAL, INCREASED ELASTIC AND OBSTRUCTIVE WORK LOAD (FIG. 11.16)

If the total WOB is plotted against respiratory frequency, there is optimal frequency at which the total work is minimal. This supports the opinion by many clinicians that the ventilatory pattern in patients with lung

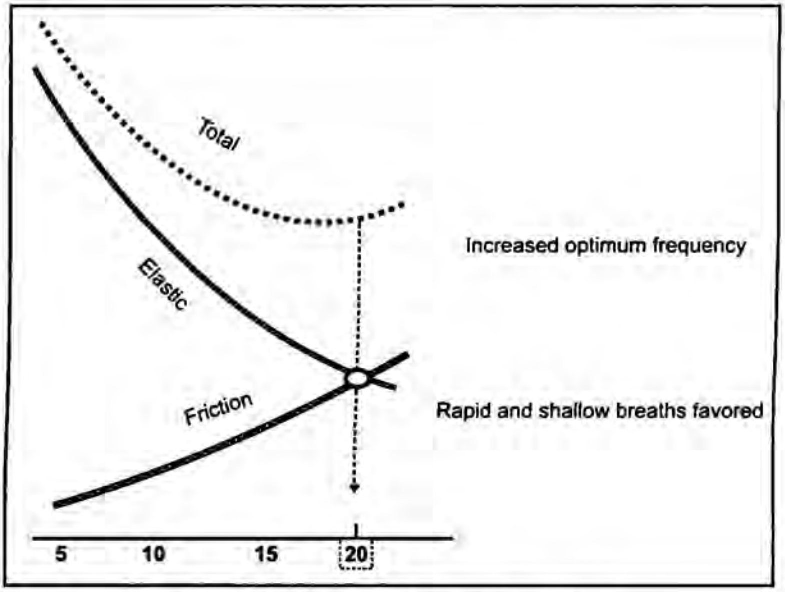


Figure 11.14: Increased elastic component

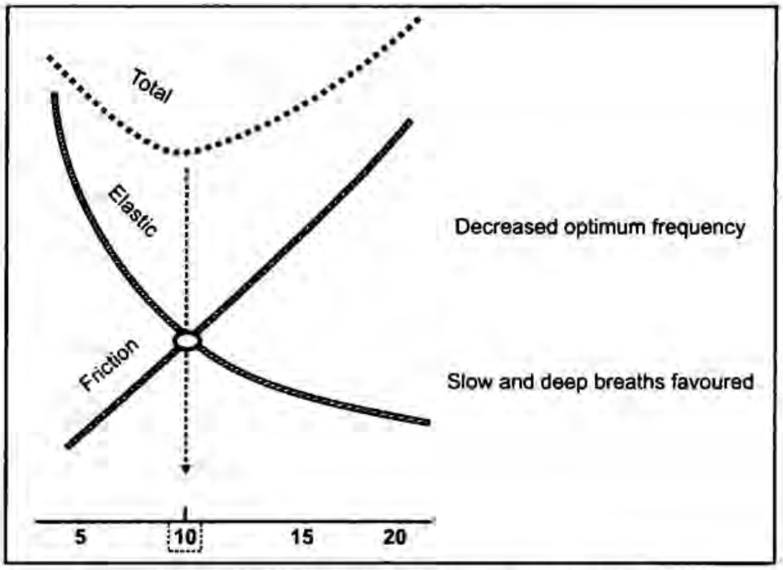


Figure 11.15: Increased obstructive component

diseases is determined entirely by the minimum expenditure of energy and not by the most physiological ventilation in terms of optimal gas exchange.

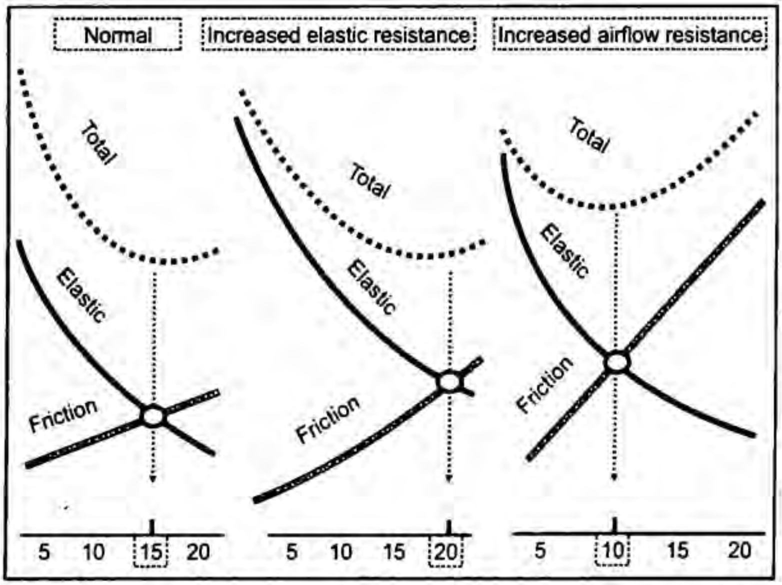


Figure 11.16: Comparison of normal, increased elastic and obstructive work load (For colour version *see* Plate 5)

Added Work Imposed by the Ventilator (Fig. 11.17)

The following will increase the work of breathing:

- Breathing through an ETT.
- A ventilator which is difficult to trigger or a ventilator with only pressure triggering (this requires more WOB than flow triggering).
- A too low flow than required by the patient.
- An increased expiratory resistance.

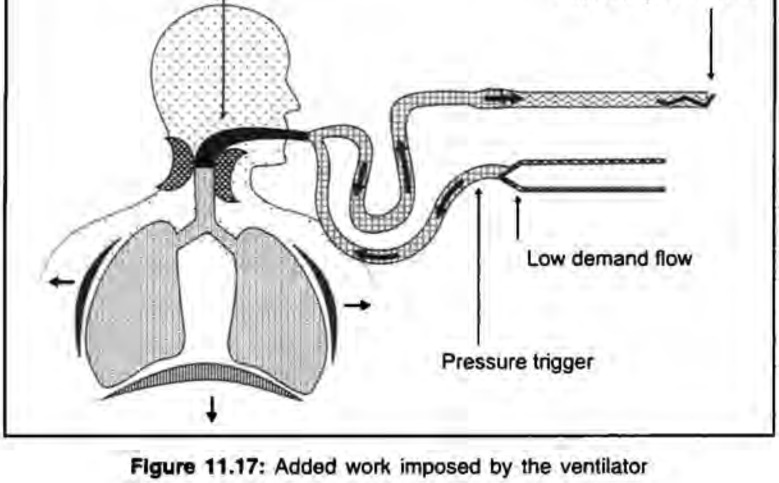


Figure 11.17: Added work imposed by the ventilator

PSV as a mode of assisted ventilation may be advantageous in decreasing the patient's work of breathing and improving the patient's comfort.

CLINICAL SIGNS OF INCREASED WOB

Careful clinical observation can give useful information on increased WOB.

- Respiratory rate and minute volume
- Chest and abdominal wall movement
- Accessory respiratory muscle activities
- Other signs.

Respiratory Rate and Minute Volume

Minute volume is the product of tidal volume and respiratory rate.

$$\mathbf{MV = V_T \times f}$$

A given minute volume may be the result of a large tidal volume and a slow rate or a small tidal volume and a rapid rate. Each level of minute volume has an optimal respiratory rate at which WOB is minimal.

Respiratory Rate in Normal Person (Fig. 11.18)

The adult or infant patient will select respiratory rate to improve the efficiency of breathing depending on the type of mechanical problem.

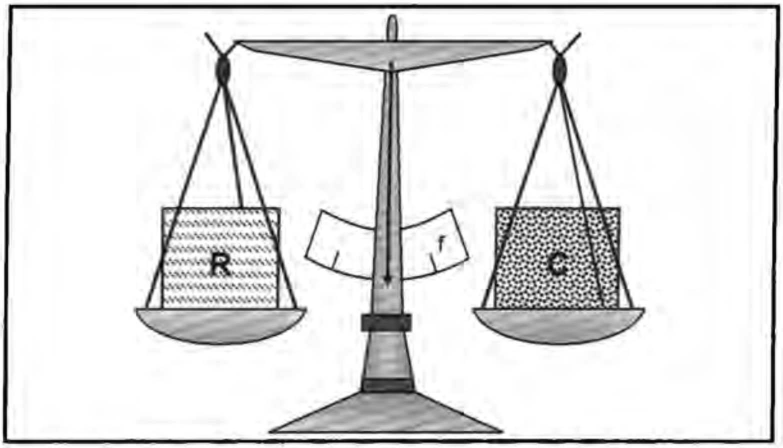


Figure 11.18: Respiratory rate in normal person

A small tidal volume means minimal elastic resistance and a low rate means minimal air way resistance. Indeed, it is common knowledge that patients with restrictive lung disease adopt a pattern of ventilation that combines small tidal volumes with a high rate while those with chronic obstructive pulmonary disease (COPD) adopt a large volume-low rate pattern. Be aware that an increased rate (above 30 for the adult and above 60 for the infant) is a clinical sign of respiratory muscle fatigue.

Patients with Decreased Compliance (Fig. 11.19)

If the compliance is low e.g. postoperative (low lung volume or abdominal distension) or ARDS/RDS breathing will be rapid and shallow.

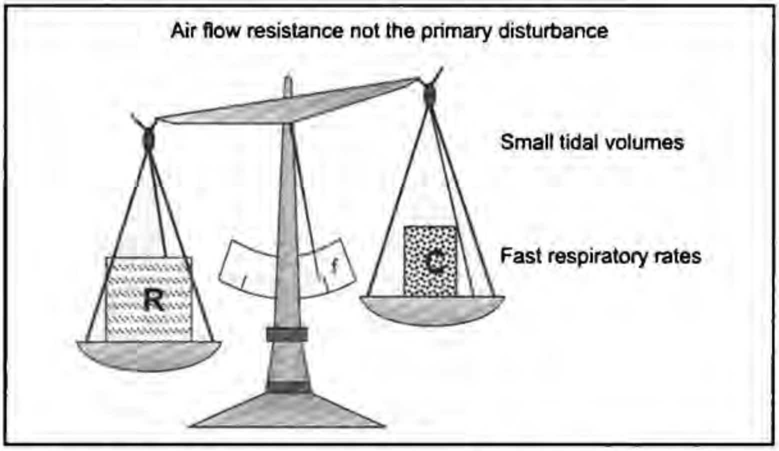


Figure 11.19: Respiratory rate in patients with decreased compliance

Conversely, at high rates (coupled with small tidal volumes), airway resistance is the more important determinant of WOB.

Patients with Increased Resistance (Fig. 11.20)

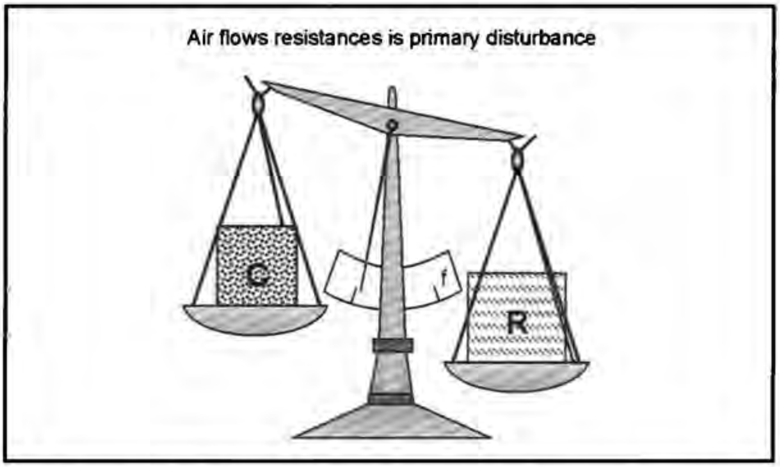


Figure 11.20: Respiratory rate in patients with increased resistance

If resistance is increased, e.g. small tube, airway obstruction (asthma, bronchitis, emphysema) breathing will be slow and deep. A large tidal volume-low rate combination results in the elastic component being the predominant determinant of WOB as at low rates airway resistance is minimal.

Chest and Abdominal Wall Movement

Respiratory alternans (sequential use of diaphragm followed by the other inspiratory muscles) can be seen in an in-patient with increased WOB and is predictive of respiratory muscle fatigue.

Accessory Respiratory Muscle Activities

This can be easily evaluated by palpitation of the sternocleidomastoid muscle, in the neck. During contraction it becomes tense. Some patients, typically a patient with end stage emphysema, show chronic diaphragmatic failure and rely solely on the accessory muscles.

Other Signs Include

- Nasal flaring
- Tissue retractions typically seen in the supraclavicular region and at the supraclavicular notch
- In infants it will show intercostal retractions.

PART-II

Effects of Controlled Ventilation

CHAPTER

12

Physiological Effects of Spontaneous v/s Controlled Respiration

ROLES OF VARIOUS RESPIRATORY PRESSURES IN VENTILATION

There are several physiological effects of controlled ventilation on various organ systems. Some of the effects are harmful. Mechanical ventilation will increase oxygen transport but will decrease cardiac output nullifying its beneficial effects. To minimize the harmful effects of controlled ventilation, we have to understand the difference between spontaneous and controlled respiration. It is vital to understand the role of various respiratory pressures in ventilation which will help to decide the ventilatory modes and strategies.

Transairway Pressure (Fig. 12.1)

In quiet, unobstructed respiration, even at the height of gas flow, the pressure difference across the airway is small. During quiet breathing it is –1 cm H_2O at the peak of inspiration and +1 cmH_2O at the peak of expiration. This pressure difference across the airway is called transairway pressure, which is comparatively of a small magnitude since it has to overcome only the airway resistance.

Transthoracic Pressures (Transalveolar pressure + Transpleural pressure) (Fig. 12.1)

The relationship between the volume and the pressure of the gas in the alveoli, when both are measured as changes from their resting levels, is determined by the elastic properties of the lungs and the chest wall. The main effort of the respiratory muscles being used to overcome the elasticity of the lungs. The difference between the alveolar and intrapleural pressure overcomes the elasticity of the lungs (Transalveolar pressure). The difference between the pleural and atmospheric pressure overcomes the elasticity of the chest (Transpleural pressure). Pressure difference between the atmospheric pressure and the alveoli is larger, ranging from –10 cmH_2O at the end of inspiration to –5 cm H_2O at the end of expiration. The magnitude of these two pressure differences (exterior and the airway – transairway and alveolar and atmospheric pressure – transthoracic pressure) differs from each other.

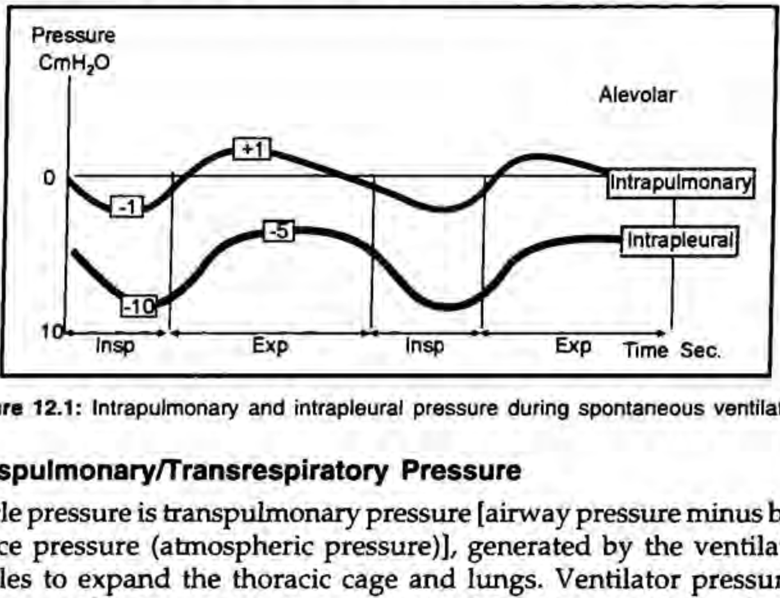


Figure 12.1: Intrapulmonary and intrapleural pressure during spontaneous ventilation

Transpulmonary/Transrespiratory Pressure

Muscle pressure is transpulmonary pressure [airway pressure minus body surface pressure (atmospheric pressure)], generated by the ventilatory muscles to expand the thoracic cage and lungs. Ventilator pressure is the transpulmonary pressure generated by the ventilator during inspiration.

Transpulmonary pressure is needed to drive gas to alveolar level and distend the lung and chest wall. The total inflation pressure (transpulmonary pressure) during positive pressure ventilation is composed of three primary elements. It is the combination of transairway pressure and transthoracic pressure (Transalveolar + Transpleural Pressure). Air flows from the outside (atmosphere) to the inside of the lungs because there is a pressure difference created between the outside and the alveoli.

- Since the pressure at the mouth is atmospheric, the pressure in the alveoli during inspiration must be subatmospheric for air to flow.
- By the end of inspiration the pressure within the alveoli has become atmospheric again. When expiration starts, the pressure in the alveoli raises a few cmH_2O above the atmospheric pressure and gradually falls to atmospheric again as the lungs empty.

Spontaneous Breathing—Negative Pressure Gradient

During spontaneous ventilation, the respiratory muscles, principally the diaphragm and other respiratory muscles, increase the size of the thoracic cavity. This increases intrathoracic volume and a fall in intrathoracic pressure within the thorax secondary to it. The intrapleural pressure falls from end-expiratory level of –5 cm H_2O to –10 cm H_2O at the height of inspiration causing the alveolar pressure to become negative relative to atmospheric pressure. With each inspiration, airway and pleural pressure falls below atmospheric pressure and gas flow is delivered to the lungs under a negative pressure gradient.

Relationship of atmospheric pressure (P_{atm}) and alveolar pressure (P_A) during spontaneous breathing

Spontaneous breathing	P_{atm} cmH_2O	P_A cmH_2O	ΔP	Flow
Inspiration	0	– 5	– 5	Into lungs
End-inspiration	0	0	0	None
Expiration	0	+ 5	+5	Out of lungs
End-expiration	0	0	0	None

Positive Pressure Breathing—Positive Pressure Gradient

During positive pressure breathing, airway pressure increases above atmospheric pressure and gas flow is delivered to the lungs due to positive pressure gradient.

Role of Transpulmonary Pressure in Determining Tidal Volume (Fig. 12.2)

The difference between airway pressure (atmospheric pressure in spontaneous ventilation) and pleural pressure is called transpleural pressure, which determines tidal volume. During mechanical ventilation the degree by which the increase in airway pressure is transmitted to the pleural space is determined by the lung and chest wall compliance.

$$\text{Lung compliance} = \frac{\text{Tidal volume}}{\text{Change in transalveolar pressure}}$$

$$\text{Chest wall compliance} = \frac{\text{Tidal volume}}{\text{Change in transpleural pressure}}$$

Normally lung and chest wall compliance is about equal, which implies that the transpulmonary pressure equals the change in pleural pressure.

Lung compliance = Chest compliance

Transpulmonary pressure = Change in pleural pressure

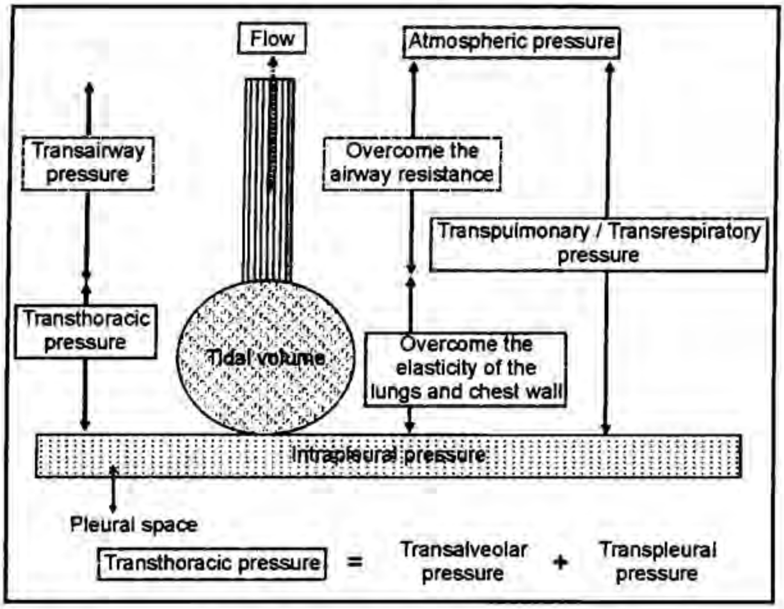

Flow
Atmospheric pressure
Transairway pressure
Overcome the airway resistance
Transpulmonary / Transrespiratory pressure
Transthoracic pressure
Tidal volume
Overcome the elasticity of the lungs and chest wall
Intrapleural pressure
Pleural space
Transthoracic pressure = Transalveolar pressure + Transpleural pressure

Figure 12.2: Respiratory mechanics

As the transpulmonary pressure also equals the difference between the airway and pleural pressure in normal lungs, it is obvious that about one-half of the increase in airway pressure is transmitted to the pleural space, the heart and the great vessels within the chest. The dominant cardiovascular effect from mechanical ventilation is a decrease in venous return, which in turn depends greatly on the magnitude of the change in pleural pressure. The changes in the pleural pressure during a mechanical breath thus depends on tidal volume and lung and chest wall compliance

In the presence of high tidal volume, high lung compliance or low chest wall compliance, the increase of pleural pressure will be exaggerated. That is why in patients with emphysema (high lung compliance), circular bandage around the chest or large chest burns; low chest wall compliance is more likely to develop cardiovascular depression. Low tidal volume or low lung or high chest wall compliance minimizes changes in pleural pressure. Patients with adult respiratory syndrome, congestive heart failure (low lung compliance) or an open chest after median sternotomy (high chest wall compliance) is relatively more resistant to the effects of cardiovascular depression.

Controlled Ventilation – Positive Pressure Breathing (Fig. 12.3)

During positive pressure ventilation, gas flow is delivered to the lungs under positive pressure gradient (i.e. airway pressure is always greater than alveolar pressure during inspiration). When positive pressure is used for ventilation, the pressure in the airways, alveoli and pleura is increased during inspiration.

Intrapulmonary Pressure

The pressure which is present in the lungs is called intrapulmonary or alveolar pressure. During controlled respiration with positive pressure alone, the pressure in the alveoli rises from the atmospheric depending upon the type of mode and lung mechanics. During the expiratory phase the pressure falls to atmospheric as the lungs empty.

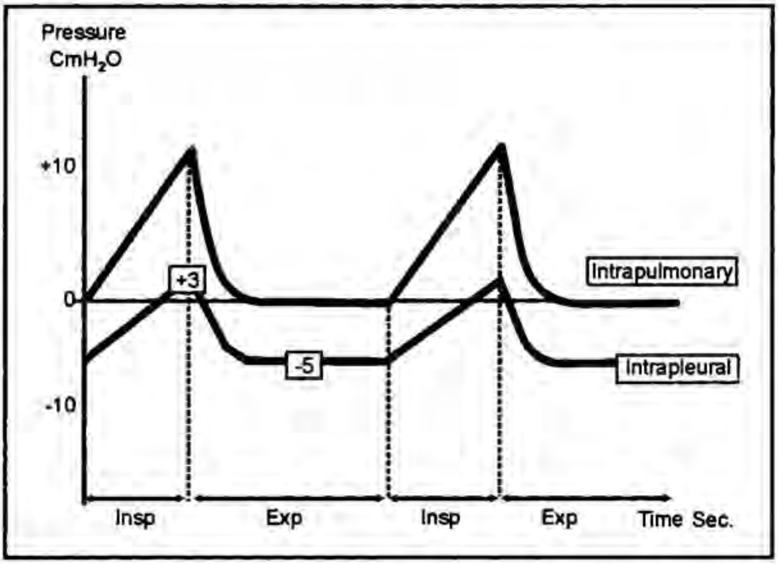


Figure 12.3: Intrapulmonary and intrapleural pressure during controlled ventilation

Intrapleural Pressure

In controlled respiration, under the same conditions, the intrapleural pressure rises during the inspiratory phase from –5 cmH_2O to +3 cm H_2O, falling to – 5 cmH_2O again during expiration.

Relationship of inspiratory pressure (P_I) and alveolar pressure (P_A) during PPV

PPV	*P_I cmH_2O*	*P_A cmH_2O*	*ΔP*	*Flow*
Inspiration	15	0	+ 15	Into lungs
End-inspiration	15	15	0	None
Expiration	0	15	+ 15	Out of lungs
End-expiration	0	0	0	None

To summarize the major difference between spontaneous breathing and positive pressure breathing lies in the intrathoracic pressures during inspiration.

ANAESTHETIC AND CLINICAL APPLICATIONS OF DIFFERENCES OF VENTILATION IN TERMS OF VENTILATION AND PERFUSION

The Spontaneously Breathing Supine Patient (Fig. 12.4)

Spontaneous ventilation provides optimal distribution of ventilation in relation to perfusion.

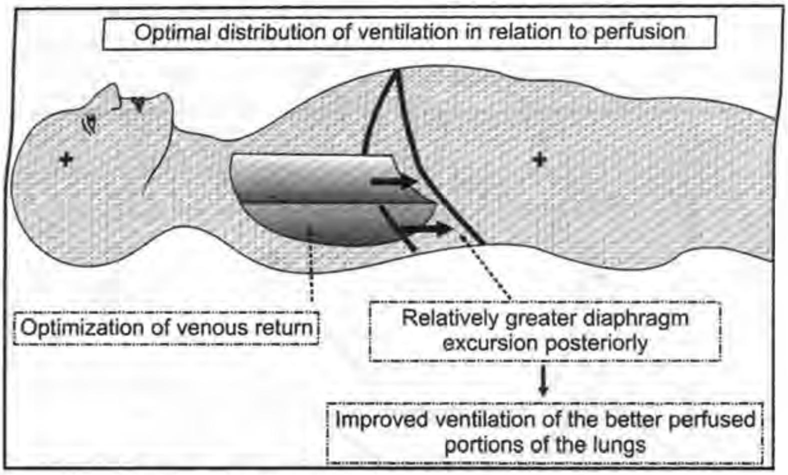

Optimal distribution of ventilation in relation to perfusion
+
+
Optimization of venous return
Relatively greater diaphragm excursion posteriorly
Improved ventilation of the better perfused portions of the lungs

Figure 12.4: Spontaneously breathing supine patient

Optimized Venous Return

Venous return is optimized in spontaneously breathing patients because intra-abdominal and intracranial pressures are more positive than intrathoracic pressure which is negative. The dependent part of the lung is better perfused due since blood flow is gravity dependent.

Improved Ventilation Posteriorly

The diaphragm has greater excursion posteriorly than anteriorly. This allows improved ventilation of the better perfused portions of the lungs.

Controlled Ventilation (Fig. 12.5)

Positive pressure ventilation results in the following:

1. It favors pulmonary blood flow toward the gravity dependent portions of the lung

   Positive pressure ventilation potentially hinders venous return because intrathoracic pressure is more positive than intra-abdominal or intracranial pressure.

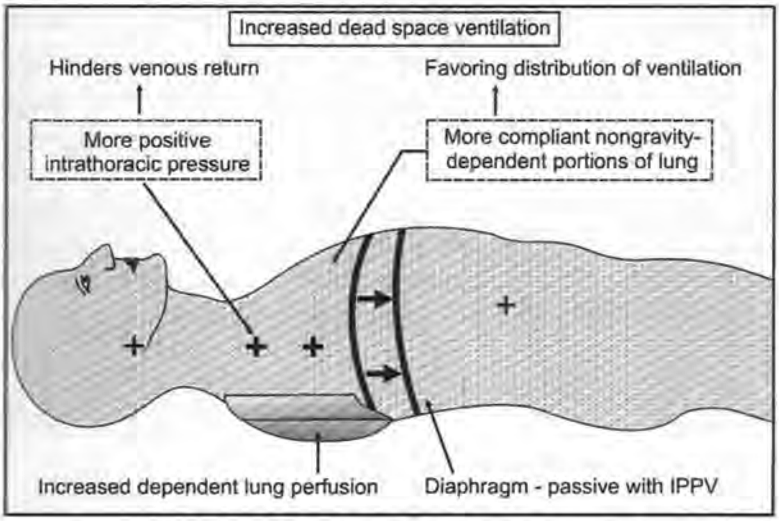

Increased dead space ventilation
Hinders venous return
Favoring distribution of ventilation
More positive intrathoracic pressure
More compliant nongravity-dependent portions of lung
Increased dependent lung perfusion
Diaphragm - passive with IPPV

Figure 12.5: Controlled ventilation in supine patient

Positive pressure breathing:

- *Directly increases mean airway pressure.*
  The increased alveolar pressure with IPPV tends to augment the normal distribution of pulmonary blood flow towards the gravity dependent portions of the lung.
- *Indirectly increases intrathoracic pressure.*
  The elevated intrathoracic pressure increases right atrial pressure which will diminish blood flow to the right ventricle. The resultant decrease in pulmonary perfusion favors pulmonary blood flow toward the gravity dependent portions of the lung.

2. Greater air distribution to the non-gravity-dependent portions of the lungs
   Ventilation is favored to the non-gravity dependent lungs during positive pressure ventilation. This is explained primarily by the attenuation of two physiologic mechanisms associated with spontaneous breathing.
   - *Decreased pleural pressures*
   - *Diaphragm function*
     In controlled ventilation the diaphragm is essentially passive, favoring distribution of ventilation to the more compliant, non-gravity-dependent portions of the lungs.

This combination of increased dependent lung perfusion and non-dependent lung ventilation with IPPV results in increased dead space ventilation (ventilation in excess of perfusion) even when adequate cardiac output is maintained.

CLINICAL IMPORTANCE OF RESPIRATORY CHANGES

The above changes can give rise to worsening of blood gases after the patient is put on the ventilator. During IPPV instead of improving, blood gases may worsen. One of the reasons for these abnormal blood gas values, directly caused by IPPV, is inadequate expansion of vascular volume leading to diminished cardiac output giving rise to hypoxaemia (decreased $S\bar{v}O_2$ and increased dead space ventilation). Assuming the cardiac output is adequately supported, the most important effect of IPPV on the V/Q is increased dead space ventilation.

CHAPTER

13

Harmful Effects of Controlled Ventilation

Valsalva recognized the connection between the venous return and the intrathoracic pressure in the 17th century. Valsalva's experiment was to try to expire against a closed glottis, which raises the intrathoracic pressure to a positive level which interferes with the venous return and causes marked distension of the peripheral veins, especially in the head and neck. The blood pressure falls and if the experiment continues, the subject can lose consciousness as a result of cerebral ischemia.

HARMFUL EFFECTS OF CONTROLLED VENTILATION

1. Cardiovascular effects
    - Abolition of the thoracic pump mechanism
    - Cardiac performance
        - Preload
        - Afterload
    - Role of CVP
2. Respiratory system
    - Damage to lungs
        - Volutrauma
        - Barotrauma
    - Uneven ventilation
3. Decreased perfusion
    - Kidneys, liver, and gastrointestinal
4. Neurological effects.

The heart, great vessels and the pulmonary vascular bed lie within the chest. Any change in intrathoracic pressure which is associated with mechanical ventilation will affect directly these structures. It is mandatory to understand the effects of PPV on the various systems to minimize the damage.

CARDIOVASCULAR SYSTEM

Factors Influencing Cardiovascular System

- Intrathoracic pressure – most important
- Changes in lung volume

1. Intrathoracic Pressure

- Spontaneous ventilation – Negative pressure ventilation
  The fall in the intrapulmonary pressure within the thorax during spontaneous ventilation not only sucks air into the lungs but also blood from outside the thorax into the great thoracic veins and the heart.
- Controlled ventilation – Positive pressure ventilation (Fig. 13.1)
  The abolition of the **'negative thoracic pump'** mechanism is the main factor for the cardiovascular effects. Mechanical ventilation, by modifying subatmospheric pressure, disturbs this important mechanism. Positive pressure within the lungs is transmitted to the structures within the thorax.

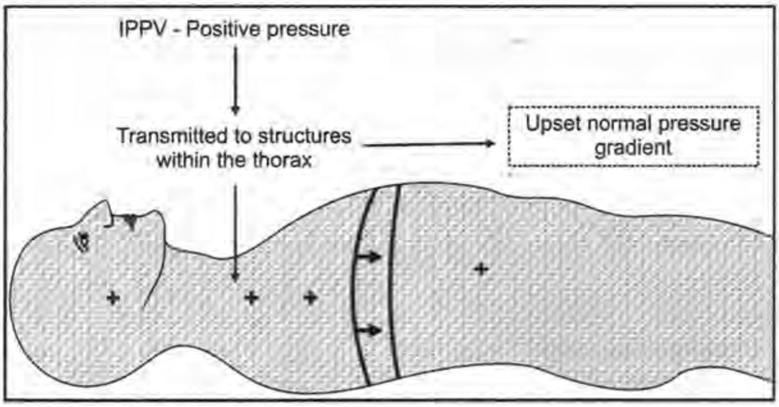

IPPV - Positive pressure
Transmitted to structures within the thorax
Upset normal pressure gradient
+
+
+
+

Figure 13.1: Abolition of the negative thoracic pump mechanism

Due to this the normal pressure gradient between the intrathoracic veins and those outside is upset. It decreases venous return resulting in a fall in blood pressure when the patient is put on the ventilator.

2. Intrapleural Pressure (Fig. 13.2)

- During spontaneous breathing the intrapleural pressure is negative and at the height of inspiration it is around –10 cm H_2O. But during controlled respiration this becomes + 3 cm H_2O.

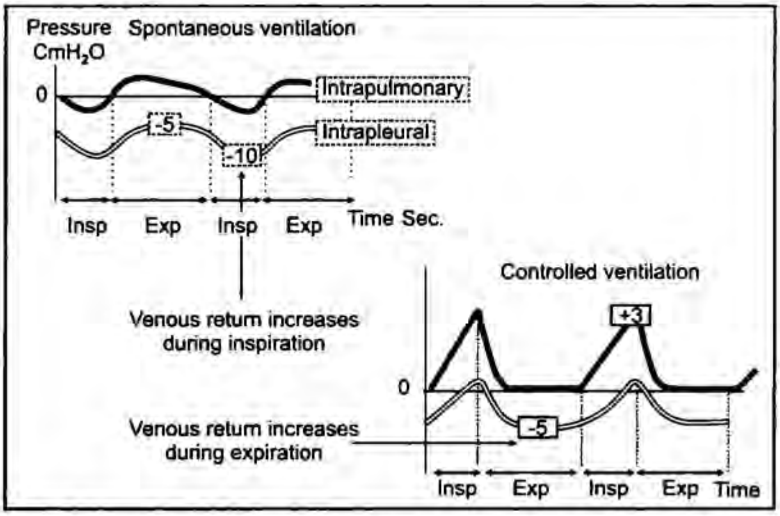


Figure 13.2: Effects of intrapleural pressure on venous return

- During spontaneous and controlled respiration, intrapleural pressure is always negative during the expiratory phase.
- During spontaneous breathing the venous return is greatest during inspiration and in controlled respiration during the expiratory phase.

The most important circulatory effect of mechanical ventilation is to reduce venous return as intrathoracic/intrapulmonary pressure rises during a mechanical breath.

Influence of Positive Pressure Lung Inflation on Heart

Positive-pressure lung inflation can influence the heart in various ways

1. Cardiac performance
   - Preload
   - Afterload
   - Contractility
2. Pulsus paradoxus
3. Role of CVP.

Cardiac Performance

(a) Influence of intrathoracic pressure on transmural pressure (stretch)
The influence of positive pressure ventilation on cardiac performance involves change which produces in preload and afterload for both

the sides of the heart. To understand these changes it is important to understand the influence of intrathoracic pressure on transmural pressure. It is the pressure that determines ventricular filling (preload) and the resistance to ventricular emptying (afterload).

(b) Alveolar-capillary units showing the transmission of alveolar pressure (P_{alv}) to the pulmonary capillaries (Fig. 13.3)

P_c = Capillary hydrostatic pressure
P_{tm} = Transmural pressure across the capillary wall
V_T = Tidal volume delivered by the ventilator

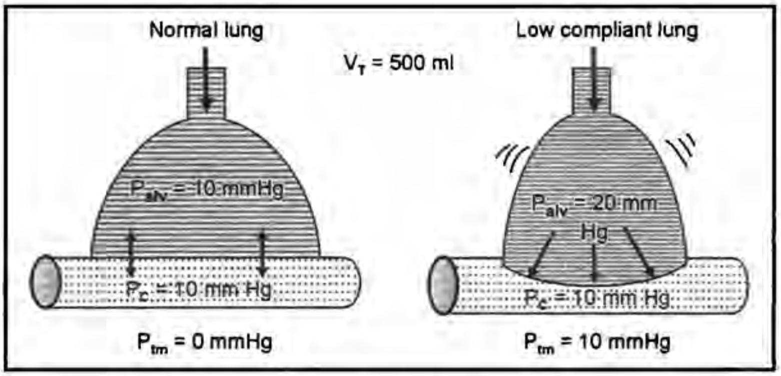


Figure 13.3: Alveolar-capillary units showing the transmission of alveolar pressure (P_{alv}) to the pulmonary capillaries

- *Normal lung*

  When a normal lung is inflated with 500 ml from positive-pressure source, the increases in alveolar pressure are completely transmitted into the pulmonary capillaries and the transmural pressure is zero.
- *Low compliant lung*

  When the same lung inflation occurs in lungs that are not easily distended the increase in alveolar pressure is not completely transmitted into the capillaries and the transmural pressure increases.

(c) Effects of increase in transmural pressure

This increase in transmural pressure acts to compress the capillaries. Therefore, in conditions associated with a decrease in lung compliance (e.g. pulmonary edema, pneumonia, ARDS), positive-pressure lung inflation tends to compress the heart and intrathoracic blood vessels.

Preload

Preload, which is the length of the cardiac muscle fibres at the beginning of contraction, is estimated by the amount of blood present in the ventricle at end-diastole (end-diastolic volume).

The mechanism whereby mechanical ventilation can decrease ventricular filling – preload (Fig. 13.4).

I. Positive intrathoracic pressure decrease the pressure gradient for venous inflow into the thorax.
   1. During inspiration
      - *Decreases preload of the right ventricle:*
        The preload of the right ventricle (i.e. the venous return) decreases as vena caval and right atrial pressure increases and blood is hindered from entering the chest.
      - *Increases preload of the left ventricle:*
        The preload of the left ventricle increases as the pulmonary vascular bed is squeezed due to an increase in the intrathoracic pressure. Due to squeezing, blood flow increases through the pulmonary vein, increasing blood flow to the left atrium and the left ventricle.
      - *Disparity between outputs of both the ventricle:*
        Positive pressure breath gives rise to disparity between outputs of both the ventricles. The right ventricle output decreases and the left ventricle output increases.

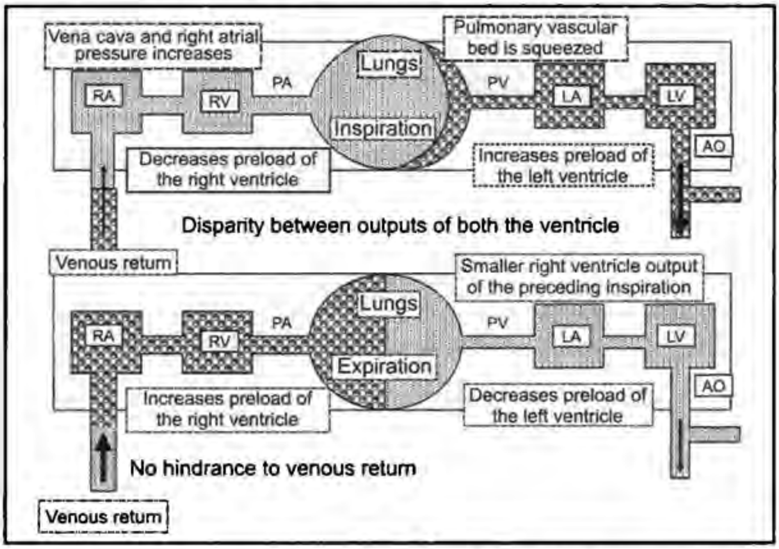

Vena cava and right atrial pressure increases
Pulmonary vascular bed is squeezed
Lungs
RA
RV
PA
PV
LA
LV
Inspiration
AO
Decreases preload of the right ventricle
Increases preload of the left ventricle
Disparity between outputs of both the ventricle
Venous return
Smaller right ventricle output of the preceding inspiration
Lungs
RA
RV
PA
PV
LA
LV
Expiration
AO
Increases preload of the right ventricle
Decreases preload of the left ventricle
No hindrance to venous return
Venous return

Figure 13.4: Circulation showing the effects of positive pressure breath on the preloads of the right and left ventricle (For colour version *see* Plate 6)

2. During expiration
   Intrathoracic pressure returns to end-expiratory value.
   - *Increases preload of the right ventricle:*
     Intrathoracic pressure returns to end-expiratory value and the preload of the right side increases since there is no hindrance to venous return.
   - *Decreases preload of the left ventricle:*
     Preload to the left side decreases as a comparatively smaller right ventricle output of the preceding inspiration.
   - *Disparity between outputs of both the ventricles:*
     During late inspiration and early expiration, right ventricular output becomes greater than that of the left ventricle.

II. Any increase in positive pressure on the outer surface of the ventricles will reduce ventricular distensibility (Fig. 13.5).
Ventricular distensibility in turn reducing left ventricular filling during diastole.

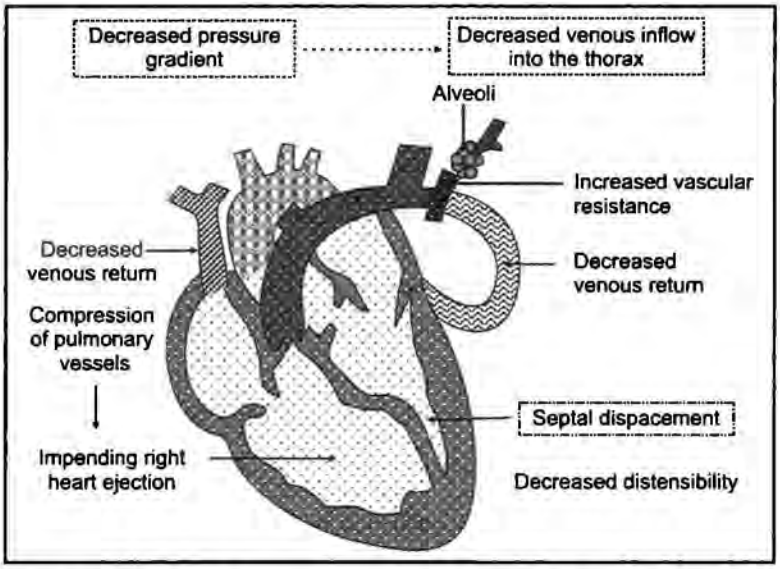

Decreased pressure gradient
Decreased venous inflow into the thorax
Alveoli
Increased vascular resistance
Decreased venous return
Decreased venous return
Compression of pulmonary vessels
Septal dispacement
Impending right heart ejection
Decreased distensibility

Figure 13.5: Positive pressure lung inflation can reduce ventricular filling

III. Positive pressure breath compresses pulmonary blood vessels. Compression of pulmonary vessels can impend right heart ejection and can dilate right ventricle. The right ventricle dilation pushes interventricular septum towards the left ventricle and reduces left ventricular chamber size. Right heart failure can impair the performance of the left side of the heart called **'ventricular interdependence'** (Fig. 13.6).

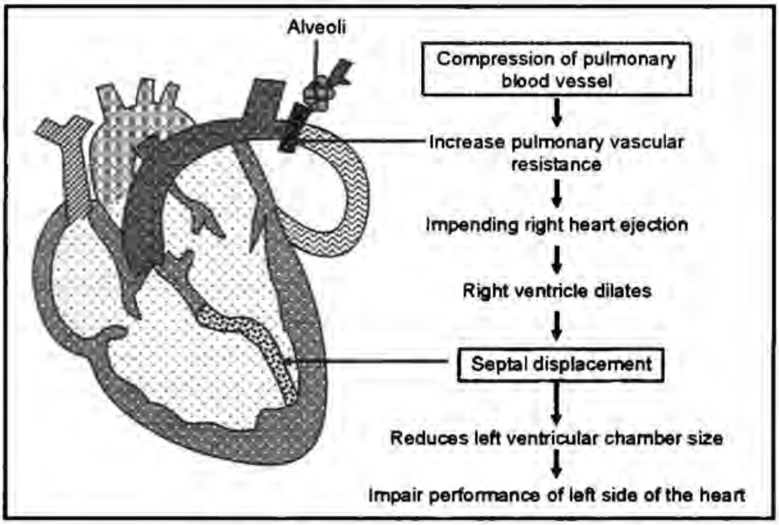

Alveoli
Compression of pulmonary blood vessel
Increase pulmonary vascular resistance
Impending right heart ejection
Right ventricle dilates
Septal displacement
Reduces left ventricular chamber size
Impair performance of left side of the heart

Figure 13.6: Ventricular interdependence

Beneficial effects of decreased preload in pulmonary oedema
The reduction of venous return results in reduced pre-load (nitro-effect) which may benefit patients with pulmonary oedema by improving pulmonary congestion.

Afterload

The increase in intrathoracic pressure affects the right and left ventricle afterloads. The afterload is the myocardial tension that develops during systole, usually equated with the vascular resistance that the ventricle must overcome to eject blood.

1. *Right ventricle afterload*
   Normally the right ventricular afterload, i.e. the pulmonary vascular resistance, is minimal at resting lung volume, i.e. at functional residual capacity.
   i. *Not significant change in normal lung*
   As lung volume increases major pulmonary vessels dilate and their resistance falls while juxta-alveolar vessels are compressed by inflating alveoli and their resistance increases. The overall change in pulmonary vascular resistance during mechanical ventilation of normal lung is therefore not significant.
   ii. *Increased after load in hyperinflated lung*
   In patients with hyperinflated lungs such as COPD, asthma or high levels of PEEP, additional increase in lung volume may significantly increases right ventricular afterload due to significant increase in resistance of juxta-alveolar vessels.

iii. *Patients ventilated with a different level of PEEP*

Patients ventilated with a different level of PEEP increases pulmonary vascular resistance gradually as increase in airway pressure. Right ventricular contractility is preserved until pulmonary artery pressure rises to a critical level.

II. *Left ventricular afterload*

This is because afterload is a transmural pressure force. It is influenced by the pleural pressures at the outer surface of the heart.

i. *Increase in pleural pressure decreases afterload*

Increase in airway pressure will increase pleural pressure which is transmitted to the left ventricle and the thoracic aorta. The pressures in the left ventricle and the thoracic aorta increases transiently compared to extrathoracic aorta. Due to this increase in pressure, the tension that the left ventricle has to develop to eject blood is decreased.

ii. *Decrease in pleural pressure increases afterload*

There is significant decrease in pleural pressure associated with:

- Severely decreased lung compliance
- Severe bronchospasm
- Spontaneous ventilation during upper airway obstruction

This decrease in the pleural pressure will be transmitted to the left ventricle and the thoracic aorta. This will increase left ventricle afterload and influence cardiac performance.

III. *Positive pressure ventilation effects on cardiac output*

Positive airway and alveolar pressures increases subatmospheric pleural pressures that surround the heart and vena cava. The increased pleural pressure tends to compress the right atrium and vena cava, increasing the intravascular resistance and pressures associated with these structures. Positive pressure ventilation decreases cardiac output and arterial blood pressure. The effects of positive pressure breath on the cardiac output depend on baseline cardiovascular function.

i. *High mean airway pressure*

Due to mechanical breath there is a decrease in the cardiac output and effects are exaggerated if it is associated with high mean pressure.

ii. *Patient with hypovolaemia*

In patients with hypovolaemia, cardiac output will be influenced by any further change in the inadequate venous return and mechanical ventilation will dramatically decrease cardiac output.

In hypovolemic patients drop in cardiac output and blood pressure can occur when mechanical ventilation is instituted, especially when PEEP is used.

Positive-pressure lung inflation tends to reduce ventricular filling during diastole but enhances ventricular emptying during systole as if hand squeezing the ventricle during systole. When ventricular filling is reduced, cardiac output is similarly reduced. However, when ventricular filling is not compromised, positive - pressure lung inflation can increase cardiac stroke output.

Normal Blood Pressure due to Compensatory Mechanism

Though venous return decreases significantly during controlled ventilation, blood pressure fall may not be seen due to compensatory mechanism.

The two primary compensatory mechanisms initiated by the cardiac baroreceptors include:

1. *Rise in peripheral venous pressure*
   Normally blood pressure fall is rapidly compensated by a rise in peripheral venous pressure, which reconstitutes the venous gradient and so re-establishes the venous return to its former level. The restoration of the venous gradient is essential if an adequate cardiac output during controlled respiration is to be maintained.
2. *Increased heart rate*

Clinical significance of compensatory mechanism

Cardiac output is dependent upon the capacity to adjust the redistribution of blood between the vascular compartments by change in a vascular tone and presence of an adequate circulating blood volume. If the patient is not having pre-existing cardiovascular disease and not hypovolemic, the cardiovascular system can compensate for a small drop in venous return and can maintain cardiac output and blood pressure.

Untoward circulating effects

The untoward circulatory effects of the positive pressure in the lungs will become marked in the following conditions:-

1. *Altered vascular tone*
   Capacity to adjust the distribution of blood between the vascular compartments by changes in a vascular tone is altered.

2. *Severe hemorrhage*
   Severe hemorrhage markedly reduces the volume of circulating blood. In such a patient extensive vasoconstriction may have already occurred to counteract the hypovolemia. Further compensation in these patients is impossible and a fall in blood pressure will be seen.
3. *Inspiratory phase is unduly prolonged - Tamponade of the heart*
   During the inspiratory phase of controlled respiration the heart itself is compressed to some extent between the expanding lungs as a result of which its output suffers. The higher the peak pressure and the longer the time during which it acts the greater is the cardiac tamponade and the interference with the cardiac output.

Two simple clinical observations will conform the above effects on the circulation after initiating controlled respiration.

1. The drop rate of an intravenous infusion is seen to slow as the intrathoracic pressure increases during each inflation.
2. When the CVP line is in use, not only does the venous pressure rise, but it is also seen to fluctuate with the variations in intrathoracic pressure.

Pulsus Paradoxus

1. *Decrease in systolic blood pressure during inspiratory phase of spontaneous breathing.*
   The negative pressure surrounding the heart can impede ventricular emptying by opposing the inward displacement of the ventricular wall during systole. This action is responsible for decrease in systolic blood pressure (reflecting decrease in stroke volume) that occurs during the inspiratory phase of spontaneous breathing.
2. *Exaggerated version of the normal response*
   When the inspiratory related drop in pressure is greater than 15 mmHg, the condition is called pulsus paradoxus (which is incorrect because the response is not paradoxical, but is an exaggerated version of the normal response).
3. *Closed chest compression in cardiac arrest*
   Positive pleural pressures can promote ventricular emptying by facilitating the inward displacement of the ventricular wall during systole. Rapid and forceful rise in positive pressure surrounding the heart might also produce massage like action to expel blood from the heart and great vessels in the thorax. Positive pleural pressure swings may be responsible for the hemodynamic effects of closed chest cardiac massage.

Reverse Pulsus Paradoxus

This is the proposed explanation for the success of cough CPR, which uses forceful coughing to maintain circulatory flow in patients with ventricular tachycardia. The increase in the stroke volume causes an increase in systolic blood pressure during positive lung inflation: a phenomenon known as reverse pulsus paradoxus and it explains the beneficial actions of closed chest compression in cardiac arrest.

Central Venous Pressure

The pressure in the vena cava and right atrium are approximately equal and are collectively known as CVP. The magnitude of the increase in the CVP and the resultant decrease in venous return depends on several factors, including:

- The airway pressure - mean airway pressures
- Lung compliance
- Chest wall compliance

Clinical significance of CVP

The usually low CVP, like negative intrapulmonary pressure creates an intravascular gradient between the right atrium and the systemic venous drainage that augments venous blood return to the right atrium. If positive pressure ventilation increases the CVP, the pressure gradient between the right atrium and venous drainage will be decreased with a resultant decreased venous return to the right atrium.

If the venous return is significantly reduced, this can result in a decreased cardiac output giving rise to arterial hypotension. Higher airway pressure will more likely result in higher pleural and higher central venous pressure. It is important to note that increases in the mean airway pressure tend to depress venous return more than increases in the peak inspiratory pressure. The degree of increased pleural pressure for a given airway pressure is further affected by the patient's lung and chest wall compliance.

Effects of Lung Compliance Into the Pleural Space

A. Effects of low lung compliance

1. If the patient's lung compliance is low such as "stiff lung" then airway pressure is less readily transmitted into the pleural space due to dampening effects of the lung tissue. Therefore, in patients with low lung compliance a given airway pressure will result in a small increase in pleural pressure and a less dramatic fall in the venous return.

2. This does not mean that patients with low lung compliance cannot have a significant decrease in cardiac output due to positive pressure ventilation. These patients must also be closely monitored for potentially significant decreases in cardiac output and blood pressure.

B. Effects of high lung compliance
1. Patients with more compliant lungs, such as COPD patients, tend to more readily transmit a higher pressure into the pleural space.
2. Therefore, in these patients a given airway will tend to result in a more dramatic decrease in venous return and cardiac output.

Effects of chest compliance into the pleural space

1. The effects of chest wall compliance on the transmission of airway pressure into the pleural space are exactly opposite to the effects of lung compliance.
2. A low chest wall compliance "stiff chest wall" will tend to increase the pleural pressure more significantly for a given airway pressure than a normal chest wall compliance.
3. Conditions in which the chest wall would be less compliant than normal include the application of tight wall bandage that encircles the thorax and extensive chest wall burn injuries.

EFFECTS ON RESPIRATORY SYSTEM

Damage to the Lungs

The probability of rupture of the alveoli is very small in properly conducted controlled respiration, except in conditions such as bullous emphysematous.

BAROTRAUMA

It is the term used to describe lung tissue injury or rupture which results from alveolar overdistension. In most cases:

- Peak inspiratory pressures greater than 50 cm H_2O
- Plateau pressures greater than 30 cm H_2O
- Mean airway pressure greater than 30 cm H_2O
- PEEP greater than 10 cm H_2O may induce the development of barotrauma
- The risk of barotrauma also increases with the duration of positive pressure ventilation.

Barotrauma can occur at mean airway pressures lower than 30 cm H_2O either due to the following:

1. *Patient Susceptibility*

   COPD patients are more susceptible to barotrauma presumably due to air trapping and weakened parenchymal areas (e.g. lung blebs and bullae).

2. *Uneven Distribution of Ventilation*

   Uneven distribution may be due to the following:
   - Localized changes in elasticity of alveoli or in the patency of the small airways such as are found in asthma, chronic bronchitis and emphysema.
   - Lateral posture causes uneven ventilation by decreasing ventilation of the dependent lung.
   - Surgical retractors and packs may also cause uneven ventilation by limiting expansion of small or even extensive parts of the lungs.
   - The localized accumulation of secretions.

Volutrauma (Fig. 13.7)

Uneven distribution of ventilation may result, in patients with significant airway obstruction and lung parenchymal changes. A mechanical tidal volume tends to preferentially distribute to areas of low resistance and high compliance during the early portion of inspiration. This may result

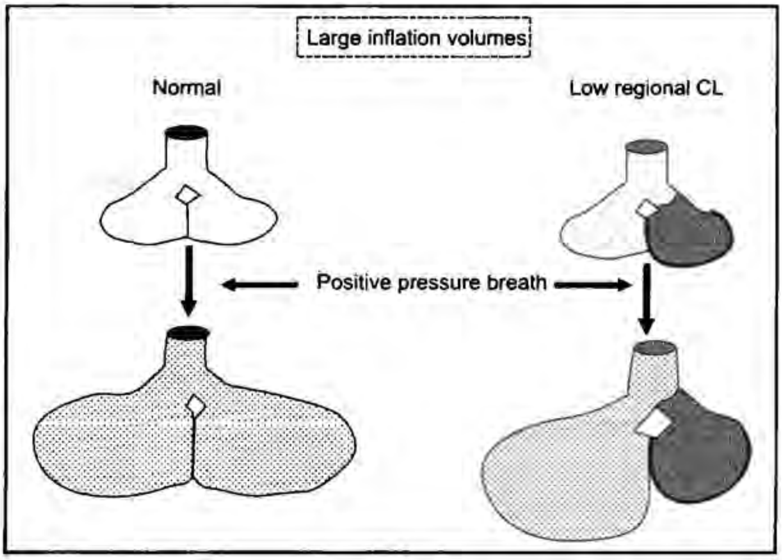


Figure 13.7: Regional compliance differences

in temporary, elevated alveolar pressure with resultant overdistension and rupture in these alveoli despite what would normally be accepted as a "safe" pressure. Because inflation volumes are distributed preferentially to regions of normal lung, they tend to over distend the normal regions of diseased lungs.

Regional compliance differences can create regions of poor inflation in diseased units while grossly overinflating healthier units during positive pressure breathing.

This tendency to over distend normal lung regions is exaggerated when large inflation volumes are used.

Hyperinflation of Normal Lungs

The hyperinflation of normal lung regions during mechanical ventilation can produce "stress fractures" in the walls of alveoli and adjacent pulmonary capillaries. These complications may be the result of excessive alveolar pressures (barotrauma) or excessive alveolar volumes (volutrauma).

Alveolar Damage

The alveolar damage can lead to alveolar rupture, with accumulation of alveolar gas in the pulmonary parenchyma (pulmonary interstitial emphysema), mediastinum (pneumomediastinum) or pleural cavity (pneumothorax, tension pneumothorax,), also pneumoperitoneum, and subcutaneous emphysema.

Rupture of the pleura in these cases is probably a secondary event, the primary process being the 'stretching away' of the alveoli from the inelastic pulmonary vascular sheaths lying between them. Subsequent breakthrough of air from the mediastinum to the intrapleural space may occur.

Damage to Pulmonary Capillaries

The damage to pulmonary capillaries can result in a leaky capillary type of pulmonary oedema.

DECREASED PERFUSION OF THE KIDNEY, HEPATIC, GASTROINTESTINAL

Effects on Kidney (Fig. 13.8)

Kidneys play an important role in eliminating waste, clearance of certain drugs and regulating fluid, electrolyte and acid base balance. The kidneys are a very vascular organ which receives 25% of the body's circulating blood volume. Due to this they are highly vulnerable to decrease in blood flow which occurs during PPV.

When renal perfusion or perfusion of the glomerular is decreased, filtration becomes less effective. The urine output is decreased as the kidneys try to correct the hypovolemic condition by retaining fluid. If hypoperfusion of the kidneys persists or worsens, renal failure may result.

Patients receiving PPV retains water and there is a decrease in serum sodium concentration and hematocrit. The greater increase in intrathoracic pressure is greater decline in renal function. The mechanism responsible for decrease in renal function is multifactorial.

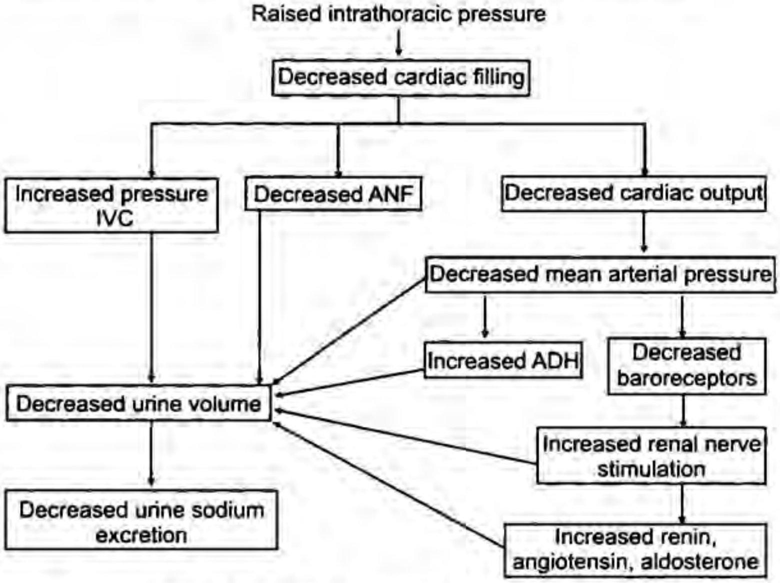

Raised intrathoracic pressure
Decreased cardiac filling
Increased pressure IVC
Decreased ANF
Decreased cardiac output
Decreased mean arterial pressure
Increased ADH
Decreased baroreceptors
Decreased urine volume
Increased renal nerve stimulation
Decreased urine sodium excretion
Increased renin, angiotensin, aldosterone

Figure 13.8: Effects of PPV on renal function

I. Direct Effects

1. *Decreased cardiac output*

   Decreased venous return to the right side of the heart, right ventricular dysfunction, alteration in left ventricular distensibility will directly decrease cardiac output. The decreased cardiac output subsequently decreases renal blood flow. This will lead to a decrease in urine output, glomerular filtration rate and urine sodium excretion.

2. *Redistribution of renal blood flow*

   Direct effect of PPV will produce changes in the intrarenal blood flow redistribution. However, redistribution does not play any significant change in renal function.

3. *Increased venous pressure*

   Increase in intrathoracic pressure increases inferior vena caval pressure which in turn increases renal venous pressure. There is a fall in renal perfusion pressure due to an increase in renal venous pressure.

II. Indirect Effects

1. *Sympathetic stimulation*

   The kidneys have autonomic innervation via the renal sympathetic nerves. These nerves are affected by changes in carotid sinus baroreceptors activity.

   Fall in mean arterial pressure decreases baroreceptor activity which results in increase in sympathetic stimulation. Renal sympathetic stimulation results in a decreased renal blood flow and renal sodium excretion.

2. *Hormonal changes:* All hormones have antidiuresis effects.

   a. Antidiuretic hormone (ADH):

      ADH acts on renal collecting tubules. ADH permits diffusion of water out of renal collecting tubules into the interstitium resulting in hyperosmolar urine excretion. In the absence of ADH the collecting ducts are impermeable to water and dilute urine is produced. At present the contribution of ADH to the decline in renal function remains unclear.

   b. Renin: Angiotensin aldosterone:

      Renal juxtaglomerular cells produce renin. Renin is released in response to renal blood flow changes, renal sympathetic stimulation and the fluid composition in the distal tubules.

      Rennin acts on angiotensinogen to release Angiotensin I. Angiotensin I is rapidly converted to Angiotensin II, a potent systemic and renal vasoconstrictor and also a major factor stimulating the release of aldosterone. Aldosterone acts on the kidney to decrease urinary sodium excretion. PPV is a potent stimulant for the renin, angiotensin, aldosterone cascade which plays a significant role in PPV induced changes in the renal function.

   c. Atrial natriuretic factor:

      This is synthesized and stored by the cardiac atrial cell and released in response to increase in atrial distension. Atrial natriuretic factor possesses potent natriuretic and diuretic properties as well as an inhibitory effect on renin and aldosterone secretion.

Conditions that increase atrial distension (congestive cardiac failure and volume loading) increase the plasma level of hormones. Atrial natriuretic factor promotes diuresis. PPV with PEEP reduced atrial transmural pressure (stretch), lowered plasma atrial natriuretic factor and decreased urine output and urine sodium excretion.

Effects on Hepatic and Gastrointestinal Functions

The mechanism PPV effects on hepatic and gastrointestinal functions are unclear but are multifactorial.

1. Decreased cardiac output.
2. Increased hepatic vascular resistance
   - Elevated venous pressure
   - Elevated intra-abdominal pressure.
   - Diaphragmatic compression.
3. Elevated bile duct pressure

Effects on Hepatic Function

Hepatic perfusion accounts for about 15% of total cardiac output. The normal liver has dual blood flow and oxygen supply. Approximately two thirds of hepatic blood flow and one half of the oxygen supply are provided by the portal vein and the remainder is derived from the hepatic artery. Any factors that decrease hepatic perfusion via either of these routes would be expected to affect hepatic function adversely.

Decreased cardiac output

PPV without PEEP was not associated with a significant fall in hepatic blood flow though there is decrease in cardiac output. PEEP induces decline in cardiac output which is responsible for a decline in the hepatic blood flow.

Increased hepatic vascular resistance

Increased hepatic vascular resistance, either arterial or portal, results in a decrease in hepatic blood flow and ischemia. Factors which increase hepatic vascular resistance

- Elevation in hepatic venous pressure
- Mechanical compression of the liver, either directly or indirectly, due to increase in intra-abdominal pressure

An elevation in hepatic venous pressure will increase hepatic vascular resistance which decreases hepatic arterial perfusion. PPV may increase hepatic venous pressure by increasing inferior vena caval pressure as a result of increased thoracic pressure. This is so-called venous back pressure.

A decrease in hepatic blood flow (hypoperfusion) and function secondary to the effects of PPV have adverse effects in critically ill patients. Medications that are cleared by the liver may accumulate and result in unanticipated adverse effects.

Liver function may be monitored by measuring the prothrombin time and bilirubin and albumin levels. Impairment of liver function will increase coagulation time (prothrombin time > 4 seconds over control) increased bilirubin > 50 mg/L, decreased albumin level < 20 g/I.

Effects on Gastrointestinal Functions

Gastrointestinal bleeding

The incidence of GI bleeding is > 40% in patients receiving PPV for more than 3 days. Gastric arterial bed does not have autoregulatory capabilities. Gastric blood flow is highly dependent on arterial pressure and venous pressure. PPV could be responsible for reduction in arterial blood pressure and increase in venous pressure. This combination results in ischemia of susceptible gastrointestinal mucosa and leads to ulceration and bleeding.

Gastrointestinal distension

In patients with decreased lung compliance PPV could lead to gastrointestinal distension and potentially to barotrauma. In these patients high airway pressure causes an inspiratory gas leak around the artificial airway.

Increased intra-abdominal pressure

An elevated IAP transmits excessive pressure across the diaphragm to the heart and great vessels. In turn, this excessive pressure leads to decreased cardiac output and decreased renal perfusion. Excessive IAP also compresses the lungs and reduces functional residual capacity. Use of PEEP on patients with elevated IAP may lead to cardiovascular, renal and pulmonary dysfunction.

NEUROLOGICAL EFFECTS

Ideally, the normal equilibrium should be maintained as being part of the optimum physiological environment. The respiratory drive is influenced through the central and peripheral chemoreceptor which responds rapidly to the levels of CO_2, H^+ and O_2 in the blood. Due to this reason, the degree of ventilation CO_2 and H^+ and O_2 oxygenation can affect the normal functioning of the brain.

Hyperventilation

Over ventilation will cause a fall in the PCO_2 and a rise in the pH, i.e. an alkalemia.

Hypoventilation

Conversely, underventilation will lead to a rise in the PCO_2 and a fall in the pH, i.e. an acidoemia. A rise in PCO_2 and fall in pH is to be avoided since it leads not only to general depression of the body, including the CNS ending in coma, but also to important undesirable effects on individual organs.

Carbon dioxide acts as a vasodilator in cerebral blood vessels. Over ventilation undoubtedly leads to cerebral vasoconstriction and state resembling anesthesia itself. The mechanism whereby over ventilation causes cerebral vasoconstriction is not completely understood, though it is to a large extent dependent on the reduction of the level of CO_2 in the blood.

- *Cerebral Tissue Hypoxia*

  If hyperventilation is prolonged, cerebral tissue hypoxia may result due to the left shift of the oxyhemoglobin curve. A left shift causes higher oxygen affinity for haemoglobin but reduced oxygen release to tissues.

- *Hypophosphataemia*

  It also produces significant hypophosphataemia because of the movement of phosphate into the cells. Hypophosphetaemia interferes with cerebral tissue metabolism by reducing ATP stores and 2, 3–DPG levels, which further increases the leftward shift of the oxyhaemoglobin curve.

- *Increased Sensitivity to Hypoxaemia*

  On the heart it increased irritability and liability to arrhythmias and ultimately to ventricular fibrillation and cardiac arrest. The sensitivity of the heart and other organs, including the liver, to hypoxia is likewise increased in the presence of a raised CO_2 level.

CHAPTER

14

Minimizing the Harmful CVS Effects of Controlled Respiration

The combination of both the magnitudes of the positive pressure and the time during which it acts are of importance in terms of 'mean pressure'. The lower the mean intrapulmonary pressure during the respiratory cycle less will be the marked cardiovascular effects.

REDUCTION OF THE MEAN INTRAPULMONARY PRESSURE

Reduction of the mean intrapulmonary pressure is most important which can be achieved by various means:

1. Positive pressure is not maintained for longer than necessary
2. Inspiration is shorter than expiration
3. Reduce mean airway pressure
4. Lungs are inflated with rapid flow of gas
5. Low expiratory resistance
6. Reduce dead space

1. *Positive pressure is not maintained for longer than necessary (Fig. 14.1):* Once the required volume has entered the lungs, expiration should be allowed to begin. In healthy lungs any additional gaseous exchange with the blood will not occur once the volume exchange has taken place, unless the inspiratory time is extremely short.

   During the inspiratory phase, plateau aids the more even distribution of the inflating gas in in-patients with pulmonary disease, but it produces circulatory embarrassment.

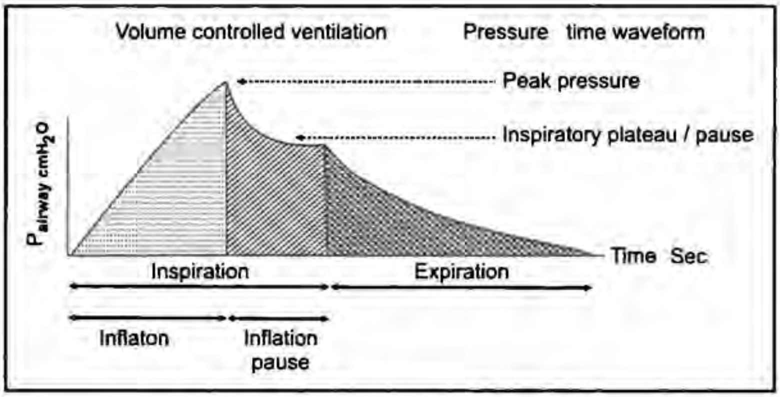


Figure 14.1: Inspiratory Pause/Plateau

2. *Inspiration is shorter than expiration - Longer expiratory phase is essential:* The expiratory phase should be more than half of the respiratory cycle. A shorter expiratory phase and longer inspiratory phase will tend to cause a reduction of cardiac output. A common setting is about 1:2. If I:E ratio is less than 1:1.5 than there will be severe circulatory embarrassment.

   An important benefit of longer expiratory phase
   - It allows the lungs to deflate which decreases intrapulmonary pressure.
   - During the expiratory phase, the heart is free from continued tamponade and its output then rises. There is reduction in intrathoracic pressure which helps to increase the venous return to great veins and the heart.

   Disadvantage of short expiratory period
   - Inflation commences while the lungs are still partially inflated giving rise to auto-PEEP.
   - The heart is subjected to continuous tamponade
   - The venous return is continuously hampered
   - Pulmonary circulation continuously hampered.

3. *Reduce mean airway pressure:*
   Even in a healthy adult the mean airway pressure within the lungs is raised, if there is a short expiratory period which will increase cardiovascular embarrassment. There will be already a degree of cardiovascular failure in chronic bronchitis and emphysematous patient. Further deterioration in cardiovascular function will be seen in case of a too-short expiratory period.

4. *The lungs are inflated with rapid flow of gas:*
   The faster the flow of gas into the lungs, shorter will be the inspiratory phase but there is a danger of alveolar rupture and uneven ventilation. But there is a tendency to use higher rather than lower flows during inflation.
5. *Low expiratory resistance:*
   Any resistance to the outflow of gas from the lungs will delay the fall of pressure during the expiratory phase and hence raise the mean intrapulmonary pressure.
6. *Reduce dead space:*
   The dead space may be reduced by the insertion of an endotracheal or tracheotomy tube and by careful design of the connections between the ventilator and the patient. By taking these steps the tidal volume needed to produce the given alveolar ventilation at a given respiratory frequency can be reduced. This will reduce the peak and hence the mean, intrapulmonary pressure.
   The harmful effects of controlled ventilation on various systems mostly depend on decreased cardiac output. By taking precautionary measures in maintaining cardiac output it will minimize the effect on various systems.

PART-III

Know Your Ventilator

CHAPTER

15

Ventilator: At a Glance

A ventilator is simply a machine or a system of related elements designed to alter, transmit and direct energy in a predetermined manner to perform useful work. Energy enters the ventilator in the form of electricity or compressed gas. This energy is transmitted or transformed (by the ventilator drive mechanism) in a predetermined manner (by the control circuit). The control circuit augments or replaces the patient's muscles in performing the work of breathing (desired output).

VENTILATOR CLASSIFICATION

Robert Chatburn (1992) has proposed a new way to classify mechanical ventilators based on related features, physics, and engineering

1. Input Power
   - Pneumatically powered - compressed gas
   - Electrically powered
     - AC
     - DC (battery)
   - Combination of both
2. Drive mechanism or Power conversion and transmission
   - External compressor
   - Internal compressor
     - Piston and cylinder (Emerson)
     - Bellows (Siemens, Servo 900 C)
     - Turbine (Bird)

- Output control valve
    - Electromagnetic poppet valve or Solenoid valve
    - Pneumatic poppet valve
    - Proportional valve
    - Microprocessor control

3. Control subsystem - Control Circuit
    - Open loop control
    - Closed loop or servo control
4. Control scheme
    - Control variables
        - Pressure controller
        - Volume controller
        - Flow controller
        - Time controller
    - Phase variables
        - Trigger variable
        - Limit variable
        - Cycle variable
        - Baseline variable
5. Conditional variable
6. Modes of ventilation
    - Breathing pattern
7. Output waveforms
    - Pressure waveforms
    - Volume waveforms
    - Flow waveforms
8. Alarm systems
    - Input power alarms
    - Control circuit alarms
    - Output alarms

INPUT POWER OR POWER SOURCE

A ventilator requires a power source. A ventilator converts the power source into a more convenient form of energy which is required to support the patient's ventilation.

Types of Input Power

- Pneumatically powered - compressed gas
- Electrically powered
- Combination of both

1. *Pneumatically Powered – Source Pressure - 50 psi*

Pneumatically powered ventilators use compressed gas as an energy source for their operation. Energy is stored as pressurized gas. The source pressure required for the ventilator to work is around 50 psi (pounds per square inch). The use of compressed gas as a power source in a ventilator is useful if no electrical power is available, such as during transport or where electrical power is undesirable, such as near magnetic resonance imaging (MRI) equipment.

Medical gases

Medical gases which are used are anhydrous and oil free. Air/oxygen is delivered at 50 pounds per square inch (psi) from a bulk gas delivery system. The source of air is through a compressor which can be central or internal. Sources of oxygen are a central pipeline, an oxygen concentrator or an oxygen cylinder.

Pressure reducing regulators

Ventilators operated by pressurized gas typically have internal pressure reducing regulators so that normal operating/working pressure is lower than the source pressure. This allows uninterrupted operation from piped gas sources in hospitals, which are usually regulated to 50 psi but are subject to periodic fluctuations. Although gases enter the ventilator at 50 psi the working pressure is usually reduced immediately to 20 psi or less. Working pressure should be as low as possible for safety purposes and to lessen tear of components.

Function of pressure

Pressure is used to inflate the lungs and also for working the control circuit. In these ventilators the gas flow required and consumed by the unit is greater than minute ventilation of the patient because ventilator gas is also consumed to power the control functions of the ventilator. To avoid insufficient supply, knowledge of this extra volume is critical when patients are being transported and pressurized gas is being provided by cylinders.

2. *Electrically Powered*

Ventilators may also be electrically powered. It utilizes either alternating current (AC) or 12 volts direct current (DC - rechargeable batteries). The AC voltage is reduced and converted to direct current DC to power electronic control circuits.

Infant and transport ventilators are designed to use rechargeable batteries as an alternative source of power when the usual AC current is not available. They are useful during transportation and for home care in case of electricity failure. Common ventilator batteries are the lead acid type with approximately 2.5 amp hours of energy. This type of battery normally requires 8 to 12 hours to recharge. The electrical power can be used to run electric motors to drive pistons, compressors or other mechanical devices like turbines and bellows that generate gas flow.

3. Combination of Both

Some ventilators are powered by a combination of both pneumatic and electric power sources. Many third generation ventilators require both an electrical power source for microprocessor control circuits and pneumatic input power source to drive inspiration. The compressed gas delivers the energy (input power) to ventilate the lungs, whereas the solenoid valves use electric power to operate the control circuit.

Gas blending—Gas mixing unit

A source of air and oxygen from the wall is introduced into a blender which can deliver a set FiO_2 in the range of 21 -100%. All critical care ventilators use either external or internal blenders. These blenders use an accurate metering device called the proportioning valve. When the oxygen port is opened, the air port is proportionately closed and vice versa. Thus, different oxygen concentrations are delivered.
In new microprocessor controlled ventilators, air and oxygen are mixed by use of a proportional solenoid valve. In some ventilators the delivered oxygen concentration is not constant. This small amount of variation may not be important clinically. The delivered oxygen concentration can be stabilized by adding a secondary external blender to ensure gas mixing before entering the ventilator. In some ventilators nitric oxide or helium can be added with air and oxygen.

Activation to provide 100% oxygen while suctioning

Activation of control provides 100% oxygen for a defined period of time (e.g. 2 minutes). It may be present in new, third generation ventilators. This is needed for hyperoxygenation immediately before and after endotracheal suctioning. In any procedure associated with desaturation, such as line insertion and postural drainage, this control can be used.

DRIVE MECHANISM OR POWER TRANSMISSION AND CONVERSION

Power transmission and conversion system is sometimes referred to as the drive mechanism. The drive mechanism generates the force necessary to deliver gas to the patient.

Compressor

A compressor is a device which is capable of building up pressure by compressing air.

Function of a Compressor

- To provide a source of compressed air.
- It draws air from the atmosphere and delivers it under pressure (50_{PSI}) so that the positive pressure breath can be generated.

Important Things to Remember

- The compressor has a filter, which should be washed with tap water daily or as directed by the manufacturer. If this is not done it greatly increases the load on the compressor.
- The indicator on the compressor should be in the green zone to work properly.
- It should not be placed too close to the wall as it may get overheated. There should be enough space to permit air circulation around it.

Types of Compressors

- *Central or external compressor*
  Some ventilators use compressed gas from wall outlets. The compressed gas through wall outlets comes from external compressors. These compressors can drive multiple ventilators.

- *Individual or internal compressor*
  Internal compressors are designed for use with a single ventilator. Turbine technology (e.g. Bird) is useful to transport the patient but may not be used long term. Turbine technology does not require compressor or compressed gas source to drive inspiration but requires electricity for the turbine to work.

Output Control Valve

This is the system used by the ventilator to transmit or convert the input power to useful ventilator work and is responsible for the characteristic output waveform. Output control valve is used to regulate the flow of gas to the patient.

- It may be a simple on/off valve called exhalation valve which may be used to shape the output waveform.
- The type of output control valve determines the characteristic flow and pressure patterns each ventilator produces.
- The use of microprocessors and proportional solenoid valves allows the newer ventilators to produce a variety of user-selected inspiratory flow or pressure patterns.

Types of Output Control Valve

1. *Electromagnetic poppet valve or solenoid valve*
   This type of device uses a magnetic force caused by an electric current to allow a small voltage to control a large pneumatic pressure in an on/off manner.
2. *Pneumatic poppet valve*
   This type of valve is similar to a solenoid valve except that it uses a small pneumatic pressure to control a large pneumatic pressure. These valves are particularly useful when electronic signals are inconvenient or hazardous. These valves are used in ventilators which use pressurized gas as a power source.
3. *Proportional valve*
   This device is similar to the solenoid valve because it is operated by an electromagnet. These solenoid valves can shape the flow waveform during inspiration by changing the diameter of its outflow port. This valve in industrial settings is called mass flow control valve.
4. *Microprocessor controlled technologies*
   Microprocessor and pneumatic technologies are combined to produce microprocessor controlled ventilators. The microprocessor is also called the central processing unit (CPU) which is the brain of controlled ventilators in which processing and decisions actually occur.

Current generation ventilators use programmed algorithms in the microprocessor to open and close the solenoid valves to mimic virtually any flow or pressure wave pattern. This is called a microprocessor-controlled pneumatic drive mechanism. Thus, microprocessor controlled ventilators are mechanical ventilators with the computing power of a microcomputer.

CONTROL SUBSYSTEMS

Control Circuit

The control circuit is the subsystem that governs or controls the drive mechanism and/or the output control valve. A control circuit assures that the breathing pattern produced by the ventilator is the one intended by the patient's care giver. This requires the setting of control parameters from the control panel.

Types of Control

- *Open Loop Control or Unspecified System (Fig. 15.1)*

  Select an input and wait for an output with no interference during the waiting period. The open loop is the simplest type of control. It cannot cope with disturbances in the system.

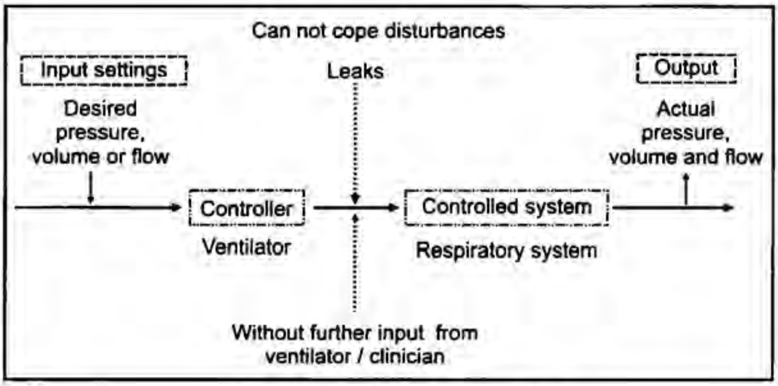

Can not cope disturbances
Input settings
Desired pressure, volume or flow
Leaks
Output
Actual pressure, volume and flow
Controller
Ventilator
Controlled system
Respiratory system
Without further input from ventilator / clinician

Figure 15.1: Open loop control

  An open loop control circuit is the one where the desired output is selected and the ventilator achieves the desired output without any further input from the clinicians or the ventilator itself.

- *Closed Loop Control or Feedback Control or Servo Controlled (Fig. 15.2)*

  Select an input; observe the trend in the output. This information from the output is used to modify the input, which in turn improves the output. This control scheme is called closed-loop control or feedback control. Feedback control is also called servo control.

  A closed loop control circuit is one where the desired output is selected and the ventilator measures a specific parameter or variable (flow, pressure or volume) continuously and the input is constantly adjusted to match the desired output. A feedback system in closed loop/double close loop consists of a sensing element called sensor, e.g. inspiratory flow sensor, expiratory flow sensor, pressure sensor.

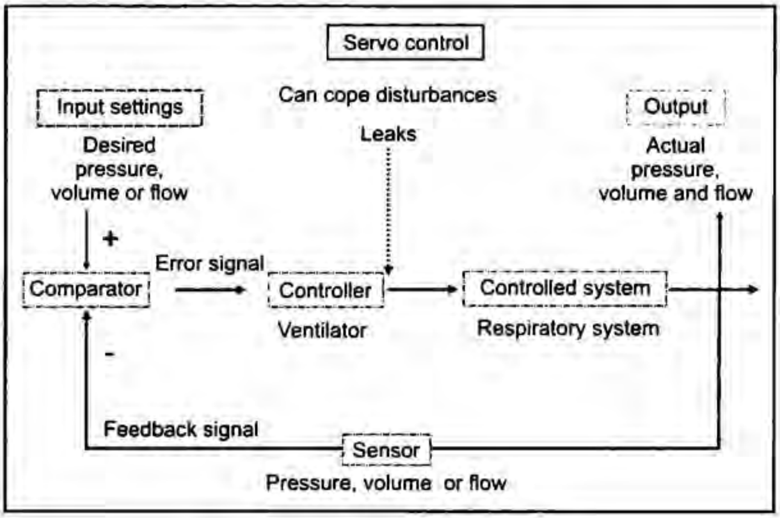


Figure 15.2: Closed loop control

- *Double-closed Loop or Dual Control*

  This system is incorporated in new ventilators to obtain advantages of both pressure controlled and volume controlled ventilation while avoiding their disadvantages.

CONTROL VARIABLES

A ventilator classification scheme based on a mathematical model known as the "equation of motion" for the respiratory system.This model indicates that during inspiration, the ventilator is able to control directly one and only one variable at a time (i.e. pressure, volume or flow).

Variables

Pressure, volume and flow are functions of time, which changes with time during the course of a breathing cycle and are called variables. These measurable variables are all measured relative to their values at end expiration.

Parameters

Elastance and resistance are assumed to remain constant and are called parameters.

$$\textbf{Pressure} = \frac{\textbf{Volume}}{\text{Compliance}} + (\textbf{Resistance} \times \text{Flow})$$

Newton's third law of motion – For every action, there is an equal and opposite reaction.

Equation of Motion (Fig. 15.3)

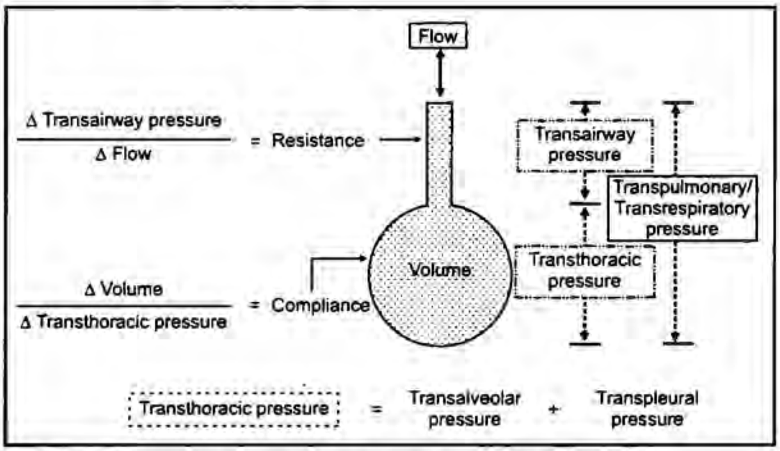

Flow
Δ Transairway pressure / Δ Flow = Resistance
Transairway pressure
Transpulmonary/ Transrespiratory pressure
Volume
Transthoracic pressure
Δ Volume / Δ Transthoracic pressure = Compliance
Transthoracic pressure = Transalveolar pressure + Transpleural pressure

Figure 15.3: Equation of motion

P_{TR} - is the transrespiratory pressure, i.e. pressure at the airway opening minus pressure at the body surface

$$P_{TR} = P_E + P_R$$

- P_E - is pressure secondary to elastic recoil - elastic load
  Elastic recoil pressure is the product of elastance (E = Δpressure Δvolume) and volume.

  Elastic recoil pressure $P_E = E \times V$ = Elastance × Volume
- P_R - is the pressure secondary to flow resistance – resistive load
  Resistive pressure is the product of resistance (R = Δ pressure/ Δ flow) and flow

  Resistive pressure $P_R = R \times \dot{V}$ = Resistance × Flow

Transrespiratory Pressure

Transrespiratory pressure can have two components

1. Secondary to ventilator –P_{vent}
2. Secondary to the respiratory muscle P_{musc}

$$P_{TR} = P_E + P_R$$

$$P_{vent} + P_{musc} = EV + R\dot{V}$$

The combined ventilator and muscle pressure causes volume and flow to be delivered to the patient.

$$\textbf{Pressure} = \frac{\textbf{Volume}}{\text{Compliance}} + (\textbf{Resistance} \times \text{Flow})$$

a. *Passive expiration*

For passive expiration, both ventilator and muscle pressures are absent. This equation becomes:

$$P_{vent} + P_{musc} = EV + R\dot{V}$$

$$- R\dot{V} = EV$$

The negative sign on the left side of the equation indicates flow in the expiratory direction. This equation also shows that passive expiratory flow is generated by the energy stored in the elastic compartment (i.e. lungs and chest wall) during inspiration.

b. *Normal spontaneous breathing*

$$P_{musc} = EV + R\dot{V}$$

$$P_{musc} = EV + R\dot{V}$$

During normal spontaneous breathing the ventilator is not needed (ventilator pressure = 0)

c. *Controlled ventilation*

$$P_{vent} + P_{musc} = EV + R\dot{V}$$

$$P_{vent} = EV + R\dot{V}$$

If the patient's respiratory muscles are not functioning, muscle pressure is zero and the ventilator must generate all the pressure for inspiration.

d. *Partial ventilatory support*

In between these two there are combinations of muscle pressure (patient effort) and ventilator pressure.

Controller

The mechanism that provides a mode of ventilation within a specific parameter (pressure, time, volume or flow) is called the controller. During ventilatory support the mechanical ventilators can control four primary variables (pressure, volume, flow and time) during inspiration. If a ventilator controls one of these variables, then that variable remains constant and its waveform does not vary with changes in compliance or resistance while waveforms of the other two variables may change.

Types of Controller

1. Pressure controller
    - Positive pressure ventilator
    - Negative pressure ventilator
2. Volume controller
3. Flow controller
4. Time controller

Pressure controller

A ventilator is classified as a pressure controller if it controls the transrespiratory pressure (airway pressure – atmospheric or body surface pressure). In pressure control mode the pressure waveform remains constant while the volume and flow waveforms may change depending on changes in compliance and resistance. This means that while you are assured that pressure will not exceed the level that has been set, the volume delivered may fall if there is a decrease in compliance or increase in resistance.

Further classification of a ventilator as positive or negative pressure ventilator depends on whether the airway pressure rises above baseline (positive) or body surface pressure is lowered below baseline (negative).

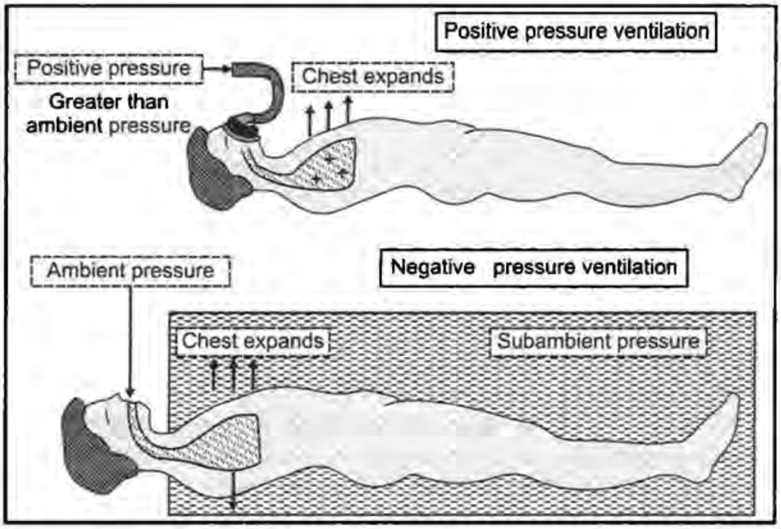

Positive pressure ventilation
Positive pressure
Greater than ambient pressure
Chest expands
Ambient pressure
Negative pressure ventilation
Chest expands
Subambient pressure

Figure 15.4: Positive and negative pressure ventilators

1. Positive pressure ventilator (Fig. 15.4)
   This type of ventilator is most commonly used today, in which airway pressure is greater than atmospheric pressure.
    - A positive pressure ventilator applies positive pressure inside the chest to expand it. Breath is delivered to the patient by closing the exhalation valve. This is maintained till preset inspiratory time.

- After the preset inspiratory time, the exhalation valve is opened and the patient is permitted to exhale passively to atmospheric pressure. Exhalation occurs because of the pressure difference between the lungs and the atmosphere and due to the elastic recoil of the lungs and the thorax.
- This type of a ventilator requires the use of a tight-fitting mask or an artificial airway.

2. Negative pressure ventilators (Fig. 15.4)
    - Negative pressure ventilators apply subatmospheric pressure outside the chest to inflate the lungs.
    - The negative pressure causes the chest wall to expand and the pressure difference between the lungs and atmosphere causes air to flow into lungs.
    - Once negative pressure is no longer applied, the patient is allowed to exhale passively to ambient pressure.

Types of negative pressure ventilator

- Tank respirator – the total body is kept in a tank except head and neck
- Body suit – this is a more "physiologic" ventilator called cuirass ventilator. The body suit is applied to the abdominal and thoracic region.

These have limited applications for patients with intrinsic lung disease, but still are of value in case of patients with neuromuscular disorders who have relatively normal lungs but impaired ventilation.

Advantages

Simplicity, reliability, ease of service and cost.

Disadvantages

- When lung disease worsens the V_T decreases and no indication is given for changes in compliance or resistance
- These machines have a small role to play in ventilating a pediatric patient with intrinsic lung disease.
- In case of a diseased lung, a ventilator cannot work due to a less pressure difference. It can work only in normal lungs. Therefore, this type of ventilator is not available now.
- A ventilator is cheap so it is more useful for prolonged ventilation such as GB Syndrome where lungs are normal, e.g. East Radcliff ventilator. It works with the help of electricity and is used in normal lungs.

Regardless of whether a ventilator is classified as positive or negative pressure, the lungs expand as a result of the positive transrespiratory pressure generated. It is the transrespiratory pressure gradient that largely determines the depth or volume of inspiration.

Volume controllers

As a volume controller, volume must be measured and used as a feedback signal to control the output, i.e. volume delivered. A volume controller allows pressure to vary with changes in resistance and compliance while volume delivery remains constant.

Flow controller

Flow controller allows pressure to vary with changes in the patient's compliance and resistance while directly measuring flow and uses the flow signal as a feedback signal to control its output.

Even though a tidal volume is set or displayed, many ventilators measure flow and then derive volume from the flow measurement

Volume (lit) = Flow (lit/sec) × Inspiratory Time (sec)

Time controller

Time controllers are ventilators that measure and control inspiratory and expiratory time. These ventilators allow pressure and volume to vary with changes in pulmonary compliance and resistance. Since neither pressure nor volume is directly measured or used as a control signal, time (inspiratory, expiratory or both) remains the only variable.

Phase Variables (Fig. 15.5)

The understanding of phase variable is useful for examining how a ventilator starts, sustains and ends an inspiration and what it does between inspirations, i.e. during expiration. Since the variable is examined during a particular phase, it is termed a phase variable. Each breath either spontaneously or mandatory goes through four phases.

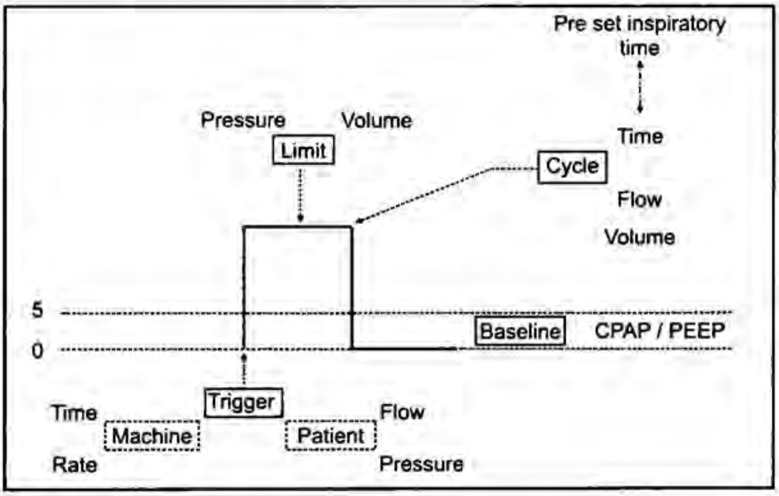


Figure 15.5: Phase variables

Mapelson proposed that ventilator-supported breath can be divided into four phases:

1. The change from expiration to inspiration—Trigger Variable
2. Variable which sustains inspiration—Limit Variable
3. The change from inspiration to expiration—Cycle Variable
4. This variable is set during expiration—Baseline Variable

1. *Trigger Variable*

The trigger variable is the variable that determines the start of inspiration. Pressure, flow or time may be measured by the ventilator and used as a variable to initiate inspiration.

- Time triggered
- Pressure triggered
- Flow triggered
- Volume triggered

Most ventilators use flow as trigger variable. Triggering should be sensitive; response should be fast i.e. 6 milliseconds.

Trigger sensitivity

- *Ventilator sensitivity*

  How hard the patient must work to initiate or trigger a breath is termed the ventilator sensitivity. If the ventilator is made more sensitive to the patient's efforts, it is easier for the patient to trigger a breath and vice versa.

- *Sensitivity level*

  The amount of negative pressure the patient must generate below the patient's baseline airway pressure (or end-expiratory pressure) or amount of flow, a patient must generate to trigger the ventilator into inspiration is the sensitivity level.

a. *Time triggered – preset time*

Time is a trigger variable when the ventilator starts a breath according to a set frequency independent of the patient's spontaneous efforts. In other words a time-triggered breath is initiated and delivered by the ventilator when a preset time interval for one complete respiratory cycle (inspiratory time and expiratory time) is complete. The respiratory rate control on the ventilator is a time – triggering mechanism.

For example, if the ventilator respiratory rate is preset at 10 breaths per minute (60 seconds), the time triggering interval for each respiratory cycle is 6 seconds. At the time trigger interval, the ventilator automatically delivers one mechanical breath every 6 seconds without regard to the patient's breathing effort or requirement (60 sec/10 breaths = 6 seconds/breath).

b. *Pressure triggered – drop in airway pressure (Fig. 15.6)*
Pressure is the trigger variable when the ventilator senses a drop in baseline pressure caused by the patient's inspiratory effort and delivers a breath independent of the set frequency.
Sensitivity or Trigger Threshold:
It is set in centimeters of H_2O relative to the baseline pressure (end expiratory pressure). A pressure triggering mechanism uses the drop in airway pressure that occurs at the beginning of a spontaneous inspiratory effort to signal the ventilator to begin inspiration.

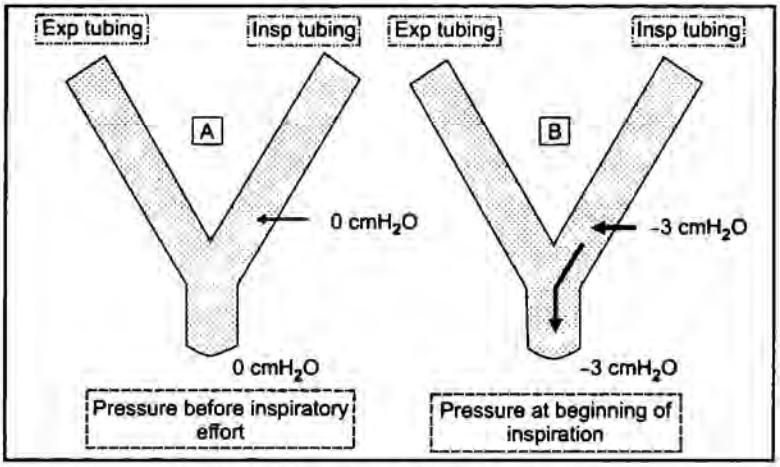


Figure 15.6: Pressure triggering mechanism

A. *Before an inspiratory effort:*
That is at the end of expiration, the pressure in the airway and ventilator tubing is equal to 0 cm H_2O. A mechanical breath is not initiated because there is no pressure drop to trigger the ventilator sensitivity settings.

B. *At the beginning of inspiration:*
The pressure in the airway and ventilator tubing is – 2 cm H_2O. A mechanical breath is initiated because the pressure drop is sufficient to trigger the ventilator sensitivity setting (assuming it is as – 2 cm H_2O or less).

If the sensitivity for pressure triggering is changed from –2 to –5 cm H_2O, the ventilator becomes less sensitive to the patient's inspiratory effort as more effort is needed to trigger the ventilator into inspiration. It means changing the sensitivity from –3 to –5 cm H_2O is decreasing sensitivity of the ventilator. The range of acceptable sensitivity levels for pressure triggering varies from –1 to –5 cm H_2O below the patient's baseline pressure.

c. *Flow or volume triggered (Fig. 15.7):*

Flow or volume is a triggering variable when the ventilator senses the patient's inspiratory effort in the form of either flow or volume into the lung. Ventilators measure flow and when the inspiratory flow reaches a specific value, a ventilator supported breath is delivered.

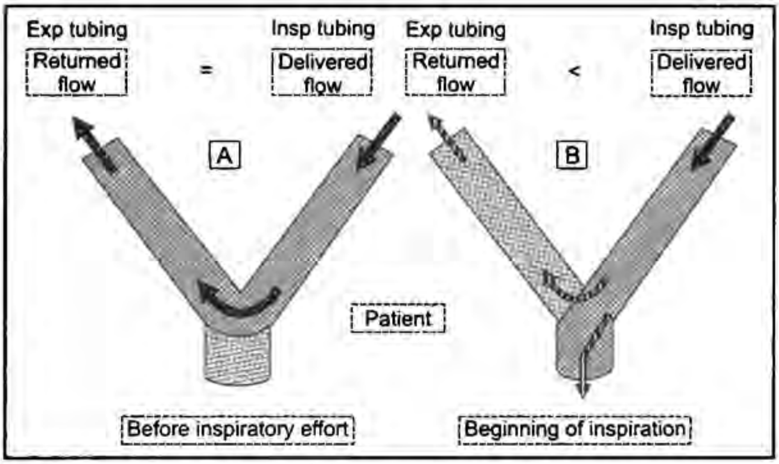


Figure 15.7: Flow trigger mechanism

A. *Before an inspiratory effort:*

That is at the end of expiration, a continuous flow passes through the ventilator circuit and returns to the ventilator (i.e., delivered flow = returned flow). Flow trigger is not activated because there is no drop in returned flow.

B. *At beginning of inspiration:*

At the start of inspiration some of the delivered flow goes to the patient due to inspiratory effort and this leads to a lower returned flow. Flow trigger is activated when the ventilator senses an inspiratory effort (i.e. the returned flow is lower than the delivered flow). The ventilator senses this flow difference and instantly supplies enough flow to satisfy the mechanical or spontaneous tidal volume. Any support breath or spontaneous breath can be flow triggered.

Flow trigger—Adult 3-6 lit, pediatric 1.5-3 lit, neonate 0.75-1.5 lit.

If the sensitivity for flow triggering is changed from 3 to 6 lit, the ventilator becomes less sensitive to the patient's inspiratory effort as more effort is needed to trigger the ventilator into inspiration. It means changing the sensitivity from 3 to 5 lit is decreasing sensitivity of the ventilator.

Advantage of flow triggering

Flow triggering has been shown to be more sensitive and responsive to a patient's effort than pressure triggering and therefore usually requires less inspiratory work than pressure triggering which helps in weaning.

d. *Inductive plethysmography*

When the infant's chest expands, a small electrical signal is generated between two chest leads, which is commonly used to monitor the infant's respiratory rate. The ventilator is interfaced to the respiratory rate monitor and inspiratory triggering occurs when the ventilator detects the signal. One infant ventilator (Sechrist IV 200) uses this inductive plethysmography to initiate a ventilator-supported breath.

This type of triggering is much faster and responsive than pressure or flow triggering since it is directly measuring the infant's inspiratory efforts.

2. *Limit Variable*

Limit means restricting the magnitude of a variable during inspiration. If one or more variables (pressure, flow or volume), is/are not allowed to rise above a preset value during the inspiratory time it is termed a limit variable. Inspiratory time is defined as the time interval between the start of inspiratory flow and beginning of expiratory flow.

- Pressure limited
- Volume limited
- Flow limited.

A limit variable does not terminate inspiration; it only sets the upper limit for pressure, volume or flow. Inspiration does not end when the variable reaches its preset value. The breath delivery continues, but the variable is held at the fixed, preset value.

3. *Cycle Variable*

The variable that is measured and used to end inspiration is called cycle variable. Inspiration ends when a specific cycle variable (pressure, volume or time) is reached. This variable is measured by the ventilator and used as a feedback signal to end inspiratory flow delivery, which then allows exhalation to begin. The mechanism which does the sensing and performs the change over is called 'cycling' mechanism of which there are four basic types:

- Time cycled
- Volume cycled
- Pressure cycled
- Flow cycled
- Mixed–New ventilator

- *Time cycled*

  In which the change over occurs after a preset time period.

- *Volume cycled*

  When a ventilator is set to volume cycle it delivers flow until a preset volume has passed through the control valve. By definition, as soon as the set volume is met, inspiratory flow stops and expiratory flow begins. If expiration does not begin immediately after the inspiratory flow stops, then inspiratory hold has been set and ventilator by definition is time cycled.

  Most ventilators measure flow and are flow controllers. Since flow is measured and used as a feedback signal for gas delivery, volume becomes a function of flow and time.

  Volume (l) = flow (l/sec) x inspiratory time (sec)

  Therefore, these ventilators are really time cycled, rather than "volume cycled". Inspiration ends because a preset time interval has passed and volume is not directly measured.

- *Pressure cycled*

  When a ventilator is set to pressure cycle, it delivers flow until a preset pressure is reached, at which inspiratory flow stops and expiratory flow begins.

- *Flow cycled*

  When a ventilator is set to flow cycle, it delivers flow until a preset level is met. Flow then stops and expiration begins. The most frequent application of flow cycling is in the pressure support mode.

- *Mixed – new ventilators*

Baseline Variable

The variable that is controlled during the expiratory phase or expiratory time is termed the baseline variable, e.g. PEEP, CPAP. Expiratory phase or expiratory time is defined as the interval between the start of expiratory flow and the beginning of inspiratory flow. Although pressure, volume or flow could serve as the baseline variable, only pressure is measured since it is more practical.

Baseline or expiratory pressure is measured and set relative to atmospheric pressure. Thus, when we want baseline pressure to equal atmospheric pressure, we set it to zero. When we want baseline pressure to exceed atmospheric pressure, we set a positive value, called positive end-expiratory pressure i.e. PEEP.

Conditional Variable

This is an important part of all modern ventilators and helps to increase the versatility and safety of these machines. Conditional variables are defined as patterns of variables that are controlled by the ventilator during the ventilator cycle. For each breath, the ventilator creates a specific pattern and specific phase variables. The ventilator either may keep this pattern constant for each breath or it may introduce other patterns, e.g. one specific pattern for mandatory and another pattern for spontaneous breath (SIMV). In other words, the ventilator decides which pattern of control and which variables to implement before each breath, depending on the value of some preset conditional variables.

Examples Include

1. Sigh – a large tidal volume breath is delivered after a specified number of breaths. This condition was incorporated in the control logic of the old ventilators.
2. Shifting from machine to patient breaths in SIMV.
3. Auto mode in Siemens 300.
4. Adaptive support ventilation in Hamilton Galileo.
5. Change from spontaneous to timed breath in BIPAP.

Early ventilators used relatively simple conditional variables (such as volume cycled, pressure limited, pressure triggered and PEEP). Newer third generation microprocessor controlled ventilators are capable of delivering complex ventilator patterns, e.g. PRVC, Auto-mode.

OUTPUT WAVEFORMS

Output waveforms are graphical representations of the control or phase variables in relation to time. Output waveforms are typically presented in the order of pressure, volume and flow. Depending on the settings, the ventilator determines the shape of the control variables, whereas the other two depends on the patient's compliance and resistance.

How to Read Waveforms?

Flow values above the horizontal axis are inspiratory, whereas flow values below the horizontal axis are expiratory. Pressure and flow values rising above the horizontal axis are for inspiration and fall back to the baseline during expiration. Careful observation and assessment of waveforms during mechanical ventilation can provide useful information to the clinician.

Waveform can assist the clinician:

- To detect inadvertent PEEP.
- In assessing the patient's ventilatory work.

Some ventilators are able to present pressure versus volume waveforms to assist in minimizing the patient's work of breathing.

- To evaluate resistance and compliance changes.
- To assess effectiveness of bronchodilator therapy.

Ventilators can present flow versus volume waveforms, which aids in the assessment of bronchodilator therapy during mechanical ventilation and the assessment of airway obstruction.

Types of Output Waveforms

- Pressure waveform
- Volume waveform
- Flow waveform

These waveforms are further classified based upon their respective shapes. Waveforms have characteristic shapes depending on the control variable.

Pressure Waveforms (Fig. 15.8)

A ventilator setting such as flow and inspiratory time, regulates how steep the waveform rises towards peak inspiratory pressure.

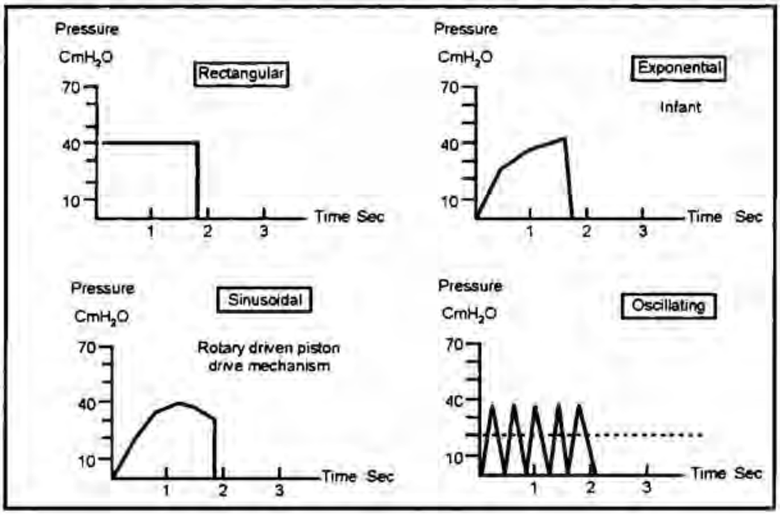


Figure 15.8: Pressure waveform

- *Rectangular pressure waveform:*
  It is a near instant rise to a peak pressure value that is maintained up to the start of exhalation. During expiration, the pressure rapidly drops to baseline.
- *Exponential pressure waveform:*
  A more gradual increase in pressure when compared with rectangular waveform. This type of waveform is common in some infant ventilators and has become an option on some adult ventilators.
- *Sinusoidal pressure waveform:*
  Sine wave is a graphic representation of flow and time that has a horizontal "s" appearance. Sinusoidal pressure waveform resembles the positive half of sine wave. It is produced by ventilators having a rotary driven piston drive mechanism.
- *Oscillating pressure waveform*

Volume Waveform (Fig. 15.9)

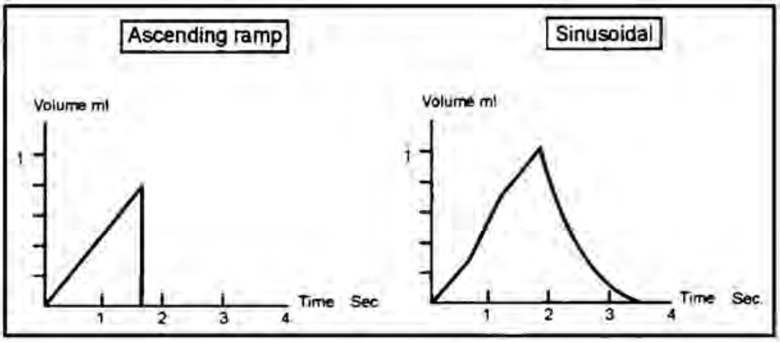


Figure 15.9: Volume waveform

- *Ascending ramp volume waveform:*
  The ascending ramp waveform is produced by a constant or rectangular inspiratory flow patterns. The shape is characterized by a linear rise to the peak inspiratory pressure value.
- *Sinusoidal volume waveforms:*
  It is produced by ventilators that have a rotary driven piston drive mechanism.

Flow Waveform (Fig. 15.10)

- *Rectangular flow waveform:*
  The rectangular flow waveform is produced when volume is the control variable. This flow waveform (a derivative of the volume waveform with respect to time) assumes the characteristic rectangular shape.

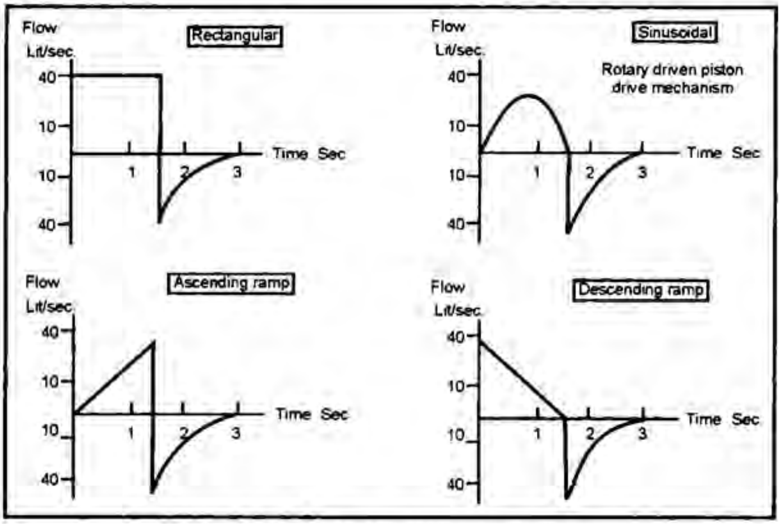


Figure 15.10: Flow waveform

- *Ramp waveform:*
  The ramp waveform can be ascending or descending. If flow rises as the breath is delivered, it is termed ascending. If flow falls during the ventilator supported breath it is called a descending ramp.
- *Sinusoidal flow waveform:*
  It resembles the positive portion of a sine wave. It is generated by a rotary driven piston drive mechanism.

Humidifier

A set FiO_2 is humidified in a humidifier and delivered to the patient by the breathing circuit. Since the endotracheal tube bypasses the normal humidifying and warming system of the upper airway, the inspired gases must be warmed and humidified.

- To prevent hypothermia
- Drying of secretions
- Necrosis of the airway mucosa.

Types of Humidifier

- Simple humidifier: It heats and humidifies inspired gas to a set temperature without a servo control. The disadvantage is excessive condensation in the tubing with reduction in the humidity along with cooling of the gases by the time they reach the patient.
- *Servo-controlled humidifier:* Servo-controlled humidifier with heated wires in the tubings. These prevent accumulation of condensate while ensuring adequate humidification.

Important Points to be Remembered

- Optimal temperature of the gases should be 36-37° and a relative humidity of 70 % at 37°C.
- If the baby is nursed in the incubator temperature monitoring must take place before the gas enters the heated field.
- At least some condensation must exist in the inspiratory limb which shows that humidification is adequate.
- The humidifier chamber must be changed daily.
- It should be adequately sterilized or disposable chambers may be used.

Breathing Circuit

Disposable Circuits

It is preferable to use disposable circuits for every patient. Disposable circuits may be changed every week.

Reusable Circuits

Reusable circuits are sterilized by gas sterilization or by immersion in 2% glutaraldehyde for 6-8 hr and then thoroughly rinsed with sterile water. If reusable circuits are used they must be changed every 3 days.

DIAGRAMMATIC REPRESENTATION SHOWING THE OPERATION OF A MICROPROCESSOR CONTROLLED VENTILATOR (FIG. 15.11)

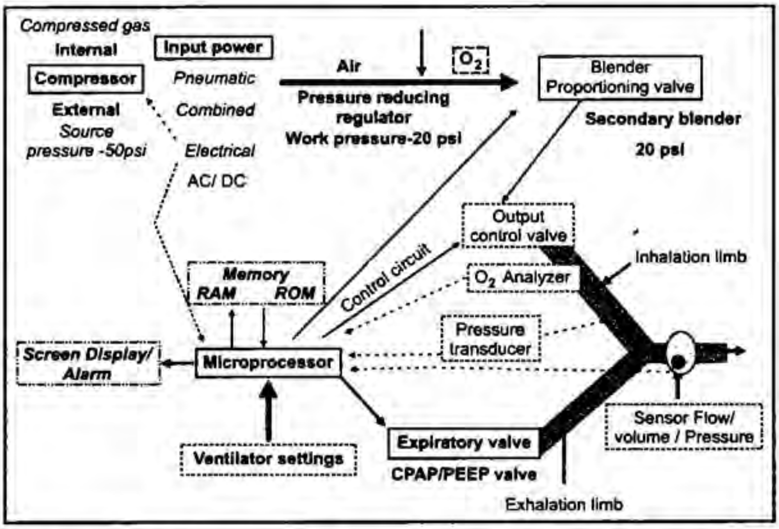


Figure 15.11: Diagrammatic representation showing the operation of a microprocessor controlled ventilator (For colour version *see* Plate 6)

Preselected ventilator settings directed to the microprocessor. The microprocessor controls the overall operation of the ventilator. The general ventilator control program can be stored on read only memory (ROM) and data is directed to the (RAM) random access memory. Feedback (dashed lines), are directed back to the microprocessor from oxygen analyzer, airway pressure transducer, and sensor (flow/volume, pressure). On the most of the ventilator sites of pressure and flow/ volume measurements are either inside the ventilator on the expiratory limb of the breathing circuit or at the y piece.

SUMMARY OF VENTILATOR FUNCTIONING (FIG. 15.12)

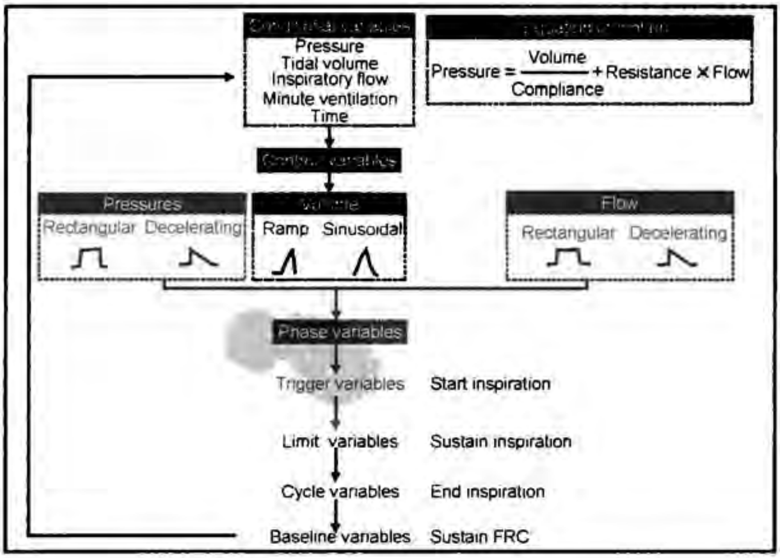


Figure 15.12: Summary of ventilator functioning

PART-IV

Ventilator Parameter

CHAPTER 16

Fractional Inspired Oxygen Concentration

Aerobic metabolism is the combustion of nutrient fuels in the presence of oxygen. The circulatory system plays a dual role in supporting this process by delivering oxygen to drive the reaction and removing the carbon dioxide that is generated. To decide appropriate settings of FiO_2, one must understand the basic features of the transport system of oxygen and the central role of haemoglobin in the transport.

OXYGEN TRANSPORT

- In the lungs
  The tension of oxygen is 105 – 110 mmHg in the alveoli (lungs) whereas it is only 40 mmHg in the venous blood entering pulmonary capillaries. This pressure gradient is sufficient to diffuse oxygen which passes across the alveolar membrane to the pulmonary capillaries.
- In the blood
  Once oxygen diffuses into the blood, it first dissolves in the plasma. Then it rapidly binds to the haemoglobin in the red blood cells for its carriage to the tissues. The binding of oxygen to an iron atom of the haemoglobin molecule is reversible. The union of oxygen and haemoglobin is broken as easily as it is formed. The affinity of haemoglobin for oxygen increases with increasing oxygen saturation Haemoglobin binds or releases oxygen depending on partial pressure of oxygen which it is exposed. In arterial blood the PaO_2 is 100 mm Hg and consequently haemoglobin is almost completely in an oxygenated form. In the tissues the blood PaO_2 drops to 40 mm Hg

and the haemoglobin is now forced to give up some of the oxygen is holding.

TOTAL OXYGEN CONTENT – CaO_2 (FIG. 16.1)

The concentration of oxygen in arterial blood is often called the oxygen content. It is the volume (ml) of oxygen present in 100 ml (dl) of blood.

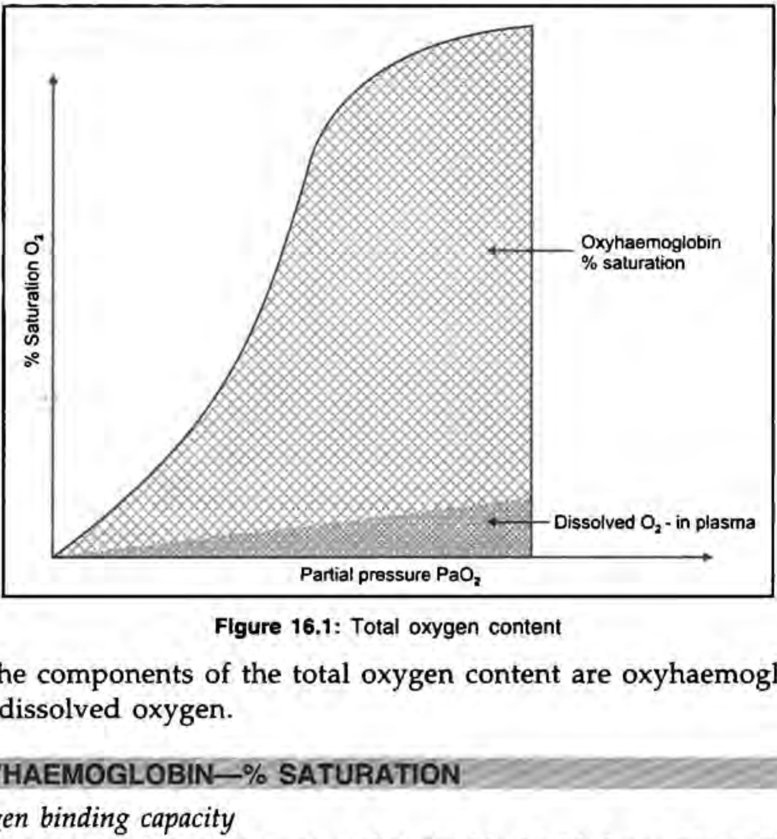


Figure 16.1: Total oxygen content

The components of the total oxygen content are oxyhaemoglobin and dissolved oxygen.

OXYHAEMOGLOBIN—% SATURATION

Oxygen binding capacity

It is the maximal oxygen amount that can bind with 1 gm haemoglobin i.e. the volume of oxygen chemically combined with haemoglobin. The relationship states that each gram of haemoglobin will bind 1.34 ml of oxygen when it is fully saturated.

Arterial Oxygen Saturation—SaO_2

$1.34 \times Hb \times SaO_2$

Haemoglobin concentration is expressed in g/dl (g/100 ml). The concentration of oxygen which is bound to haemoglobin is expressed in ml/100 ml (vol %).

$1.34\ (ml/g) \times 14\ (g/dl) \times 0.98 = 19\ ml/100\ ml$

The oxygen saturation of the blood denotes what percentages of the available haemoglobin are actually saturated with oxygen. Saturation depends on the oxygen partial pressure. In case of normal arterial pressure of oxygen of 100 mm Hg, the saturation of haemoglobin in the arterial blood is 97%.

100% saturation is not possible as a small amount of blood does not take part in pulmonary exchange, but remains as shunt blood in the arterial circulation. This short circuited blood reduces the oxygen saturation by about 3%.

Oxygen	*Mixed venous*	*Arterial*
Amount in solution in plasma	0.13 ml percent	0.3 ml percent
Tension	40 mm Hg	100 mm Hg
Amount combined with haemoglobin (Oxyhaemoglobin)	14 ml percent	19 ml percent
Saturation	75 percent	98 percent

DISSOLVED OXYGEN

Above oxygen binding capacity, oxygen will dissolve in the plasma. 0.3 ml per 100 ml of blood (0.3 volume percent) is physically dissolved in plasma. The quantity of oxygen dissolved in the plasma is calculated from the **Bunsen solubility coefficient** for oxygen in the blood.

- 0.003 ml of oxygen can be dissolved for each 1 mm Hg of oxygen tension, for each 100 ml of blood at BTPS (standard body temperature, atmospheric pressure, fully saturated with water vapor).
- A milliliter of oxygen dissolved per 100 ml of blood is expressed as volumes percent (vol %) or milliliters per deciliter (ml/dl).
- Thus 100 ml of blood with an oxygen tension of 100 mm Hg contains 0.3 ml of dissolved oxygen.

The quantity of dissolved oxygen is relatively small compared with the total oxygen carried by the blood. The volume of oxygen existing in the plasma is in the gaseous state. When the blood reaches the tissues, this small quantity is transferred to the cells. Its place is rapidly taken by more oxygen liberated from haemoglobin.

Importance of Dissolved Oxygen

- Reflects tension of oxygen in the blood, i.e partial pressure of oxygen PaO_2
- Acts as a pathway for the supply of oxygen to haemoglobin at the level of the lungs and reverse at tissue level.

HAEMOGLOBIN VERSUS PaO_2

- *Reduction in PaO_2:*
  A 50% reduction in PaO_2 (from 90 to 45 mmHg) results in only 20% decrease in CaO_2 (which is similar to the 18% decrease in SaO_2). Hypoxaemia (a decrease in PaO_2) has a relatively minor impact on arterial oxygenation if the accompanying change in SaO_2 is small.
- *Anaemia:*
  A 50% reduction in haemoglobin (from 15 to 7.5 mg/dl) is a 50% reduction in arterial oxygen content (CaO_2). Changes in haemoglobin concentration have a larger impact on arterial oxygenation than changes in PaO_2.

Anaesthetic Significance of Changes in Haemoglobin Versus PaO_2

PaO_2 influences blood oxygenation only to the extent that it influences the saturation of haemoglobin with oxygen. Therefore, SaO_2 is a more reliable index of arterial oxygenation than PaO_2.

Relative influence of anaemia and hypoxaemia on arterial oxygenation

Parameter	*Normal*	*Hypoxaemia*	*Anaemia*
PaO_2	90 mm Hg	45 mm Hg	90 mm Hg
SaO_2	98 %	80 %	98 %
Hb	15 g/100 ml	15 g/100 ml	7.5 g/100 ml
CaO_2	20 ml/100 ml	16.3 ml/100 ml	10.1 ml/100 ml
% change in CaO_2		18.6%	49.5%

OXYGEN FLUX/OXYGEN DELIVERY—DO_2 (FIG. 16.2)

Oxygen transport in arterial blood is described by Oxygen flux/oxygen delivery. Oxygen flux is the amount of oxygen leaving the left ventricle per minute in the arterial blood. Ignore the very small amount of dissolved oxygen in the physical solution.

$$\text{Oxygen flux}/O_2\ \text{Del} = Q \times CaO_2$$

$$O_2\ \text{Del} = \text{CO (ml/min)} \times \text{arterial oxygen content}$$

$$\begin{aligned} DO_2 &= \text{CO (ml/min)} \times [\text{Hb concentration (g/100 ml)} \times O_2\ \text{saturation} \times 1.34\ \text{ml/g}] \\ &= 5000\ \text{(ml/min)} \times 14.7\ \text{g/100 ml} \times 0.98 \times 1.34\ \text{ml/g} \\ &= 1000\ \text{ml/min} \end{aligned}$$

Normally about 250 ml of oxygen is used up in cellular metabolism and the rest returns to the lungs in the mixed venous blood which is therefore, about 75% saturated.

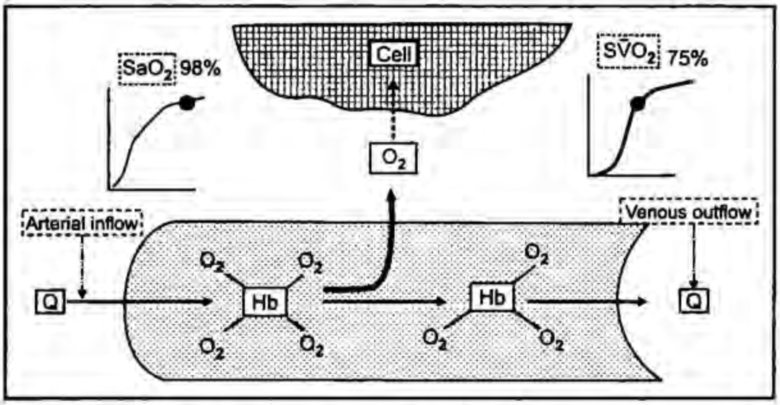


Figure 16.2: Oxygen uptake from the microcirculation

Clinical Significance of Oxygen Flux

Because the three variables in the equation are multiplied together, a relatively trivial reduction of each may result in a catastrophic reduction in oxygen flux. In addition to the reduction of the factors the oxygen consumption may be increased above 250 ml/min for e.g. in the postoperative patient suffering from halothane shakes, oxygen delivery will decrease markedly.

OXYGEN BINDING CURVE OR DISSOCIATION CURVE (FIG. 16.3)

The curve describes the relationship between the oxygen partial pressure PaO_2 and the saturation of haemoglobin (SaO_2). The curve is designed for the uptake of oxygen in the lungs and its release in the tissues.

The amount of gas which is taken up or released by plasma or haemoglobin varies according to the tension of that gas to which it is exposed. The amount that is dissolved in plasma is directly proportional to the partial pressure therefore there is linear relationship. However, the steepness of the gradient is only slight.

The iron in the haemoglobin molecule has a remarkable property of combining with oxygen to form oxyhaemoglobin. The oxygen dissociation curve or haemoglobin oxygen equilibrium curve is not linear but has S or a sigmoid shape. It has a flat lower, a steeper middle and a flat upper part. The nonlinear relationship between % saturation and PaO_2 accounts for the vast majority of oxygen reserves in the blood.

- *Vertical axis (% saturation):*

  The vertical axis represents the portion of the haemoglobin that is chemically combined with oxygen.

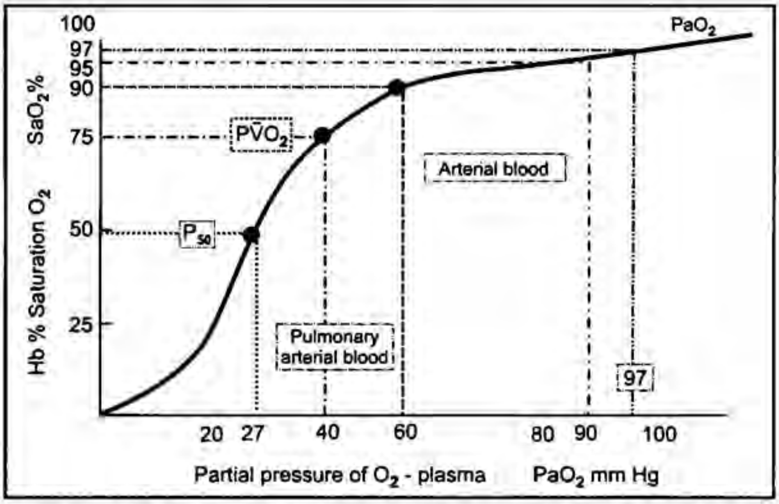


Figure 16.3: Oxygen dissociation curves (For colour version *see* Plate 7)

- *Horizontal axis (Partial pressure of oxygen):* The horizontal axis represents the partial pressure of oxygen in the plasma represented in mm Hg (kPa = mm Hg × 0.133)

For each PaO_2 there is specific oxygen saturation of haemoglobin and vice versa.

- *Normal mixed venous $P\bar{V}O_2$ (pulmonary arterial) blood*
  40 mm Hg is normally 75% saturated with oxygen
- *Arterial blood with plasma PaO_2*
  50 mm Hg is normally 85% saturated with oxygen
  60 mm Hg is normally 90% saturated with oxygen
  80 mm Hg is normally 95% saturated with oxygen
  97 mm Hg is normally 97% saturated with Oxygen

The arterial oxygen saturation, which is measured by pulse oximetry, shows small changes in the flat upper part of the oxygen binding curve that conceal large changes in the PaO_2. A drop in the oxygen saturation from 97 to 95% represents a drop of the PaO_2 from 97 to 80 mm of Hg. An oxygen saturation of 85% represents an oxygen partial pressure of about 50 mm Hg.

Relationship between the Arterial PaO_2 (mm Hg) and the Saturation of Haemoglobin

Lung-Areas with High PaO_2

During oxygen loading of haemoglobin in the lungs variation in the PaO_2 of blood leaving different portions of the lung have little effect on the oxygen content of the arterial blood because it is the flat upper part of the curve which is involved.

Blood leaving alveoli with a PaO_2 of 100 mm Hg will be 98 % saturated, where as blood leaving alveoli at a PaO_2 of 75 mm Hg will still be 95% saturated which is a drop of only 3%. In the areas with high PaO_2 the oxygen saturation increases slightly when the PaO_2 increases.

Peripheral Tissues—Areas with Low PaO_2

Peripheral tissues will be having areas of low PaO_2. Unloading of oxygen hastens when the PaO_2 falls below about 50 mmHg. This rapid release of oxygen from the haemoglobin is essential so that at capillaries PaO_2 tension is very readily available.

Here the curve takes a very steep course; even a slight decrease in the O_2 partial pressure releases a great amount of oxygen from the haemoglobin. The further the right steep part of the curve lies, the better the oxygen releasing conditions to the tissue.

Oxygen Affinity

Factors altering the affinity of haemoglobin for oxygen would change the relationship between the oxyhaemoglobin saturation and blood oxygen tension. Such alterations in haemoglobin oxygen affinity are traditionally described as shifts in the oxygen dissociation curve.

Haemoglobin carries oxygen as well as other molecules. They influence the exact position of the dissociation curve. The most important of these are carbon dioxide, hydrogen ions, and 2, 3 diphosphoglycerate (2, 3-DPG) all associated with globin. Increase in temperature, $PaCO_2$ and hydrogen ion concentration (decreased pH) shifts the curve to the right. Decrease in temperature, $PaCO_2$ and hydrogen ion concentration (increased pH) shifts the curve to the left.

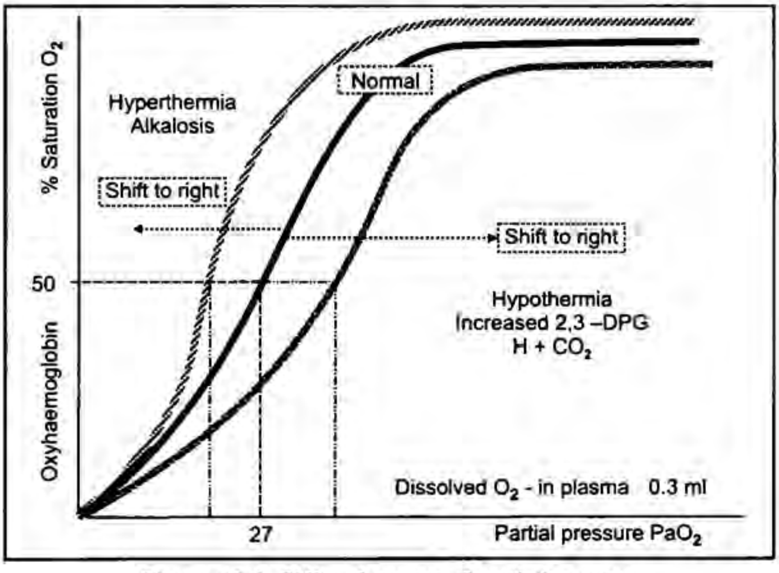


Figure 16.4: Shifts of oxygen dissociation curve

Decreased Haemoglobin Oxygen Affinity—Shift to the Right

Decreased haemoglobin oxygen affinity is most often referred to as a shift to the right, because for any given oxygen tension there is a decreased oxyhaemoglobin relative to normal. A decreased affinity should improve release of oxygen to tissues while still allowing equilibrium in the lungs. However, a significant shift to the right results in decreased oxygen content, limiting the amount of oxygen available to the tissues despite easier release to the tissues.

Increased Haemoglobin Oxygen Affinity—Shift to the Left

Increased haemoglobin oxygen affinity is most often referred to as a shift to the left, because for any given oxygen tension there is an increased oxyhaemoglobin relative to normal. The significance of a shift to the left in relation to tissue oxygenation is potentially profound because the haemoglobin is less willing to release oxygen.

For a given PaO_2 more oxygen binds to the haemoglobin, so that the oxygen saturation is correspondingly higher. Since binding between the oxygen and haemoglobin is increased, less oxygen is released from the haemoglobin.

Factors Influencing Shift of the Dissociation Curve

Carbon Dioxide Tension

Carbon dioxide acts, both, by altering the H^+ ion concentration of the solution and also by the formation of carbaminohaemoglobin which takes the place of oxygen on the haem radical. A rise in carbon dioxide tension tends to shift the curve to the right and vice versa. In the tissues the carbon dioxide tension is high, so that the haemoglobin can more readily give up its oxygen. In the pulmonary capillary blood the carbon dioxide tension falls which shifts the oxygen dissociation curve to the left which in turn helps to aid uptake of oxygen.

Hydrogen Effect—Bohr Effect

Acidaemia shifts the curve to the right and alkalaemia to the left.

2,3 Diphosphoglycerate (2, 3 DPG)

The position of the dissociation curve is related to the concentration of 2, 3 DPG within the red cells. This substance combines with globin and modifies oxygen access to the haem chain.

A rise in 2,3 DPG is associated with a reduction in the affinity of haemoglobin for oxygen. Therefore, a high concentration 2, 3 DPG shifts the curve to the right and low concentration shifts it to the left. All types of chronic hypoxia raises the level of 2, 3 DPG and storage of blood lowers it.

P_{50}

It is defined as partial pressure of oxygen (PaO_2) at which haemoglobin is 50 % saturated with oxygen when the blood is at 37⁰C, $PaCO_2$ 40 mmHg and has a pH of 7.40. The normal value for the P_{50} is 27 mm Hg.

Shifts in the dissociation curve of haemoglobin are usually presented as change in the P_{50} value. A shift to the left lowers the P_{50} and shift to the right raises it. Haemoglobin can more readily give up its oxygen when curve shift to the right and aid uptake of oxygen when curve shifts to the left.

A low P_{50} decreases the oxygen availability to the tissues and may lead to cellular hypoxia. The main consequence of alterations in P_{50} being on the release of oxygen to the tissues but have only modest effect on the uptake of oxygen in the lungs. In terms of oxygen supply of the body tissue acidosis is by far more beneficial than alkalosis. Therefore, no buffering with Na bicarbonate at $pH \geq 7.2$ is advisable.

Oxygen Supply in Tissues

Primary determination of tissue oxygen delivery (O_2 Del), i.e. the oxygen supply in the tissues depends on three factors:

- Cardiac output (CO)
- Haemoglobin (Hb) concentration
- Haemoglobin oxygen saturation

O_2 Del = CO (ml/min) × arterial oxygen content
= CO (ml/min) × (Hb concentration (g/dl) × O_2 saturation × 1.34)

If one of the three variables is reduced by half, then the oxygen supply will also be reduced by half. If all three dimensions fall to half of their original size simultaneously, the oxygen supply is reduced to one eighth. This is an oxygen amount no longer compatible with life, as the minimum consumption by metabolism amounts to 250 ml oxygen per minute.

Clinical Significance

1. Increasing the FiO_2 alters only one of this mechanism i.e. HbO_2 saturation.
2. Once the arterial oxygen tension (PaO_2) is increased sufficiently to fully saturate haemoglobin, only small additional amounts of O_2 dissolve in the plasma with additional increases in PaO_2.

Depending on the causes which reduces one of the three dimensions one refers to:

1. Stagnation hypoxia (reduced perfusion)
2. Anaemic hypoxia
3. Hypoxic hypoxia or respiratory hypoxia

The above forms rarely occur as separate entities, but one of the three causes dominates. In cases of stagnation hypoxia the problem is not reduced oxygen uptake but reduced tissue perfusion due to cardiac output or shock. Therefore the treatment of shock is also the treatment for cellular hypoxia.

Importance of O_2

1. O_2 is crucial in the production of high-energy stores (ATP) through oxidative phosphorylation.
2. Severely hypoxaemic patients supplement ATP formation via anaerobic metabolism, but this pathway is vastly less efficient than oxidative phosphorylation and it generates lactic acid.

Goal of O_2 Therapy

1. The sigmoidal shape of the O_2 saturation curve predicts that there will be little change in Hb saturation with small changes in PaO_2 at saturations >90% (corresponding approximately to a PaO_2 of (55-65 mmHg). Therefore, in normal practice, the FiO_2 or other intervention initiated to improve PaO_2 should be adjusted to maintain the Hb saturation in this range.
2. It is important to increase O_2 delivery (O_2 Del) to the tissue so that oxidative phosphorylation and other O_2 –dependent metabolic processes can continue.
3. To minimize the problem of cellular toxicity and absorption atelectasis, the lowest FiO_2 level that produces satisfactory arterial oxygenation should be used. There is no evidence that the prolonged use of a $FiO_2 < 0.40$ damages critically ill patients and therefore, no excessive effort to reduce the FiO_2 usually indicated in this range for fear of oxygen toxicity.
4. Try to maintain FiO_2 <65% to prevent O_2 toxicity.

Risk Associated with Administering High FiO_2

Potent Cellular Toxins

- *The FiO_2 level and therapy duration*

  The FiO_2 level and therapy duration associated with significant oxygen toxicity in the critically ill patient are not well defined. However, because both time and concentration are important, it is prudent to maintain the FiO_2 at 65% or lower in patients who require oxygen supplementation for an extended period.

Mechanism of oxygen toxicity

Most probably free oxygen radicals are created at high partial pressures of oxygen. If the concentration of free oxygen radicals overwhelms the lung's antioxidant defenses, cellular injury, with subsequent inflammation, oedema and pulmonary fibrosis occurs.

Absorption Atelectasis—Shunt

- If inspired O_2 concentration is high then poorly ventilated alveoli may collapse as O_2 is rapidly absorbed from the alveolus into capillary blood.
- At a FiO_2 well below 100%, these alveoli maintain their volume because of the splinting effect of nitrogen. Nitrogen exists in virtual equilibrium with nitrogen dissolved in the blood and interstitial fluids. However, at very high concentrations of FiO_2, nitrogen is washed out of the lung and O_2 becomes the predominant gas.
- The gradient between high alveolar and low pulmonary arterial PaO_2 leads to rapid absorption of this gas and instability of the alveolus unless adequate alveolar ventilation is provided. This process is termed as absorption atelectasis. This can intensify a shunt, thereby increasing hypoxemia significantly. Moreover, it may evolve in less than an hour at a FiO_2 of 100%.

Chronic Hypercapnia—Dead-space Ventilation

It can worsen CO_2 retention and respiratory acidosis.

- In spontaneously breathing patients with chronic hypercapnia high concentration of inspired O_2 can worsen CO_2 retention and respiratory acidosis. This phenomenon has been attributed to the blunting of the hypoxic drive in patients who are already not having an appropriate response to hypercapnia.
- It has been suggested that, in these patients, oxygen therapy may increase dead space ventilation and contribute to the increased hypercapnia by reducing the efficiency of CO_2 excretion.

FiO_2 Delivery

The appropriate FiO_2 delivery depends on the etiology of hypoxaemias

The pathophysiology of hypoxaemia in the acutely ill patient can be due to:

1. Hypoventilation
2. V_A/Q mismatching
3. Shunt (venous admixture)

In the Presence of Lung Pathology

1. *Hypoventilation or V_A/Q mismatching*

   Patients who are hypoxaemic secondary to hypoventilation or V_A/Q mismatching usually respond to relatively small increments of FiO_2 >21%

   - Uncomplicated overdose of sedative drugs
   - Chronic Bronchitis
   - Asthma
   - Emphysema

2. *Shunt (venous admixture)*

   An imbalance of O_2 consumption to O_2 delivery may cause mixed venous blood to desaturate abnormally, adding to the hypoxemia as the profoundly desaturated venous blood exacerbates venous admixture.

The Way to Differentiate Hypoxaemic Causes

Diagnostically, the response of PaO_2 to increase in FiO_2 can help to differentiate between hypoxemia which is due to low V/Q relationship and hypoxaemia due to true intrapulmonary shunt.

- *Relatively good response*

  In COPD the patient will have a relatively good response to FiO_2 due to low V_A/Q relationship.

- *Minimal response*

  In patients with ARDS hypoxemia is due to true intrapulmonary shunt. For a single patient, FiO_2 does influence the calculated intrapulmonary shunt. Physiologic shunt fraction varies with FiO_2 and lowest at $FiO_2 = 0.4 - 0.6$.

- *Refractory to inspired oxygen*

  Patients who are hypoxaemic from intrapulmonary shunting or intracardiac shunting are refractory to inspired oxygen and require very high O_2 depending on pathology, e.g. pulmonary edema, pneumonia, atelectasis. If the shunt fraction exceeds 40%, it is not possible to obtain an adequate PaO_2, even on a FiO_2 of 100%. In this situation, nonventilated alveoli must be recruited to reduce the shunt fraction.

Setting of FiO_2 in Various Clinical Conditions

1. For patients with mild hypoxaemia or patients with normal cardiopulmonary functions

The initial FiO_2 may be set at 40 % or at the patient's FiO_2 prior to mechanical ventilation.

- For example, drug overdose, uncomplicated postoperative recovery
- It must also be evaluated and changed accordingly by means of subsequent blood gas analysis and corrected with pulse oximeter reading.

2. Patients with severe hypoxaemia or abnormal cardiopulmonary functions

   The initial FiO_2 may be set at 100 %.
   - For example, postresuscitation, smoke inhalation, ARDS
   - The FiO_2 should be evaluated by means of arterial blood gas analysis after stabilization of the patient. It should be adjusted accordingly to maintain a PaO_2 80 to 100 mm Hg (lower for patients with chronic CO_2 retention).
   - After stabilization of the patient FiO_2 is best kept below 50% to avoid oxygen induced lung injury.
3. The disease responsible for the patient's hypoxaemia is not well defined nor is the oxygenation status known.
   - In these situations, the patient can be placed on a FiO_2 of 100% for short periods until measurement of PaO_2 are made.
   - The FiO_2 then can be adjusted to achieve Hb saturation of 90%.

Treatment of Persistent Hypoxaemia

An improvement in oxygenation may be accomplished either by increasing the inspired oxygen concentration (FiO_2) or by different ventilator settings.

VENTILATOR SETTINGS INFLUENCING OXYGENATION (FIG. 16.5)

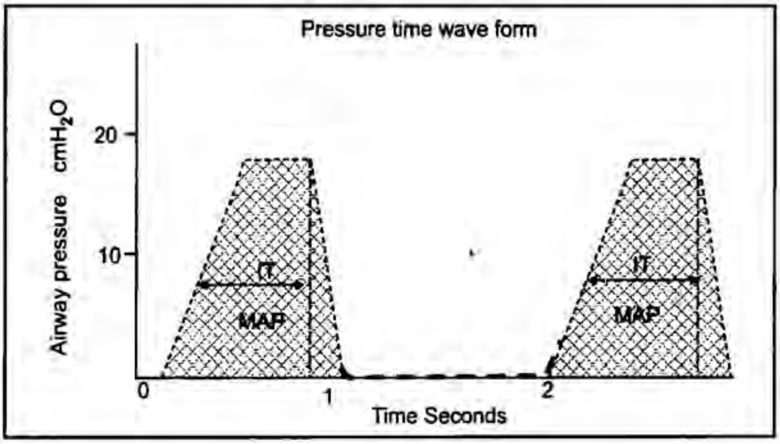


Figure 16.5: Mean airway pressure

1. In the pressure wave form MAP (mean airway pressure) is under the curve and directly affects oxygenation.
2. Oxygenation is affected by PIP, PEEP, IT and flow rate.
3. Most new ventilators digitally display MAP. If there is a need to improve the oxygenation then either MAP or FiO_2 may be increased.

Increasing any of these parameters will increase MAP and hence improve oxygenation (Fig. 16.6).

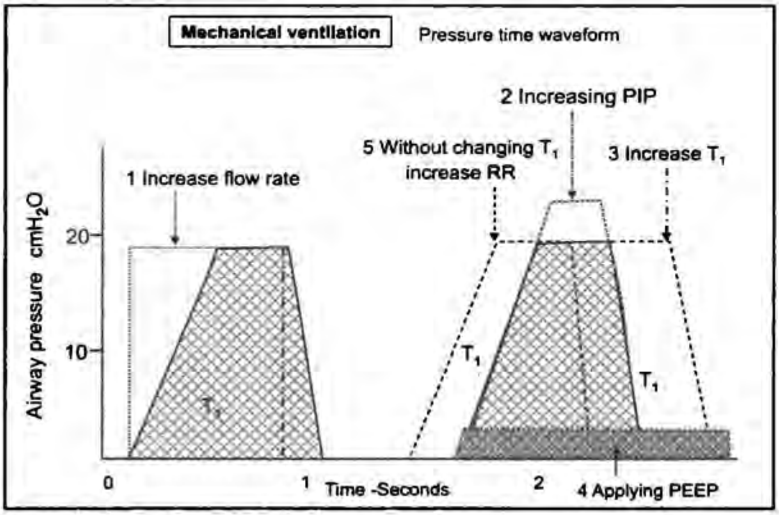


Figure 16.6: Ways to increase MAP (For colour version *see* Plate 7)

Increasing the mean alveolar pressure, may open collapsed alveoli or shift fluid out of alveoli that are flooded from pulmonary edema into the interstitial spaces.

1. Increase flow rate producing a square waveform
2. Increasing inspiratory peak pressure - PIP
3. Prolong the inspiratory time - T_1
4. Applying positive pressure in expiration - PEEP
5. Increase the ventilator rate by reducing expiratory time without changing inspiratory time.

CHAPTER

17

Tidal Volume during Mechanical Ventilation

Positive pressure ventilators expand the lungs by producing a pressure difference across the lungs and chest wall (Fig. 17.1).

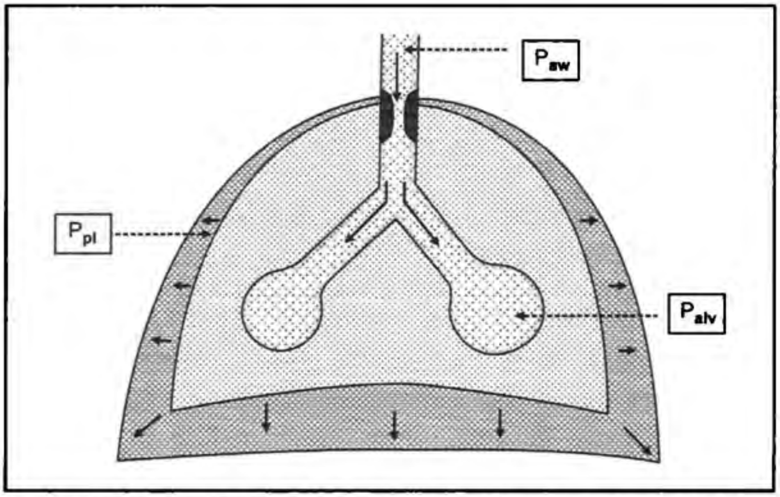


Figure 17.1: Types of pressure needed to inflate the lungs and chest

TRANSPULMONARY / TRANSRESPIRATORY PRESSURE (FIG. 17.2)

The pressure needed to drive gas to the alveolar level and distend the lung and chest wall. The total inflation pressure (transrespiratory pressure) during positive pressure ventilation is composed of three primary elements.

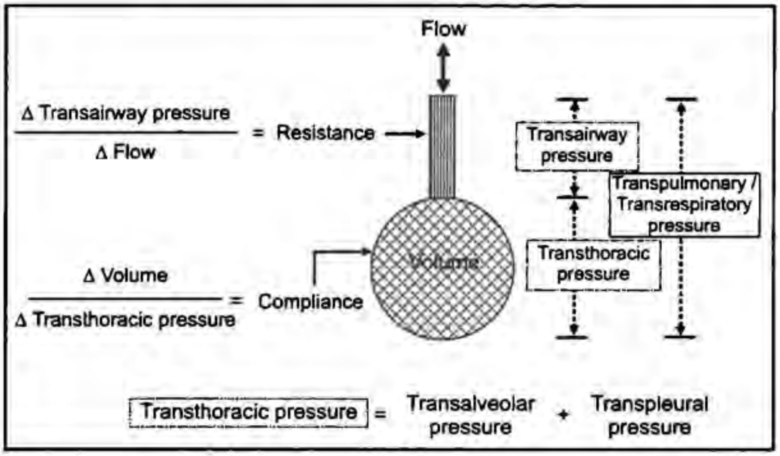


Figure 17.2: Respiratory system mechanics

Transairway Pressure

The pressure required to drive gas to the alveolar level against airway resistance (airway pressure – alveolar pressure: $P_{aw} - P_{alv}$).

Transthoracic Pressure

The pressure required to distend the lung and expand the chest wall. The pressure depends on alveolar pressure minus atmospheric pressure ($P_{alv} - P_{atm}$).

Transalveolar Pressure

The pressure needed to distend the lung against elastic forces (alveolar pressure minus pleural pressure $P_{alv} - P_{pl}$).

Transpleural Pressure (pressure across the chest wall)

The pressure needed to expand the chest wall against elastic forces is pleural pressure minus atmospheric pressure ($P_{pl} - P_{atm}$).

TIDAL VOLUME AND MODES OF VENTILATION

Tidal Volume and Pressure-controlled Ventilation

In pressure-controlled ventilators the rate of air flowing into the lung decreases, as the alveolar pressure rises with increasing alveolar volume. Flow ceases entirely when alveolar pressure equals the applied air pressure. The rate at which this equilibrium occurs is determined by the mechanical properties of the chest, i.e. airway resistance and total compliance of the lung and chest wall. The patient is not guaranteed
]]>

a constant V_T or specific flow pattern when a set level of pressure is applied to the airway.

In pressure-controlled ventilation delivered tidal volume depends on:

- *Mechanical properties of the lungs (Fig. 17.3)*
  - As compliance decreases, less volume enters the lung for any applied pressure.
  - When airway resistance increases, inspiratory airflow may decrease to the point that alveolar and airway pressures do not equilibrate at the end of the available inspiratory time; consequently, the alveoli do not reach their full equilibrium volume.
  - When compliance increases or airway resistance falls tidal volume increases for the same pressure.

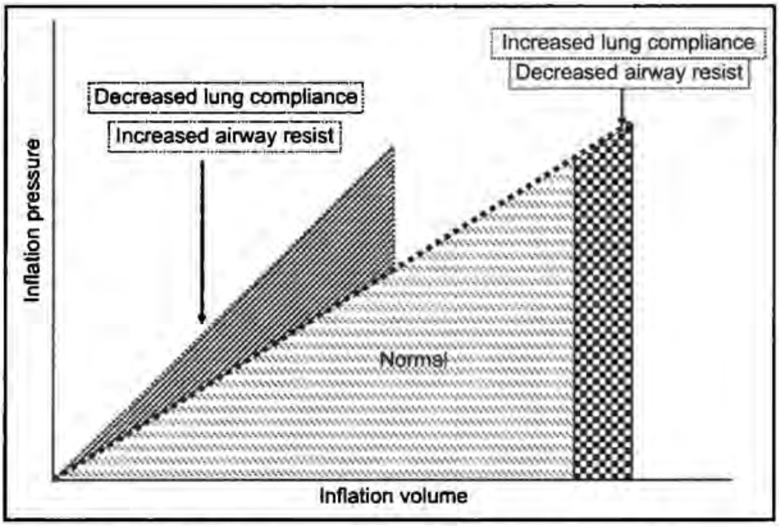


Figure 17.3: Determinants of inflation volume during PCV

- *The alveolar pressure at the beginning of inspiration*
  If there is positive pressure in the alveolus at the beginning of inspiration because of air trapping, then higher applied pressure is required to maintain V_T.

Tidal Volume and Volume-controlled Ventilation

Tidal volume is the primary parameter to be set during volume-controlled ventilation. Flow rate is the peak flow during the inspiratory phase, which determines how fast the tidal volume is delivered to the patient. Volume-controlled ventilators are flow generators which provide specified patterns of gas flow to the airway though airway pressure will vary depending on mechanical properties of the lung. The necessary pressure is generated to maintain the flow pattern depending on resistive and elastic forces during the inspiratory phase of the respiratory cycle. If lung compliance worsens, a higher ventilator pressure is required in order to deliver the same tidal volume.

Tidal Volume can be increased by increasing flow rate or inspiratory time or by increased both (Fig. 17.4).

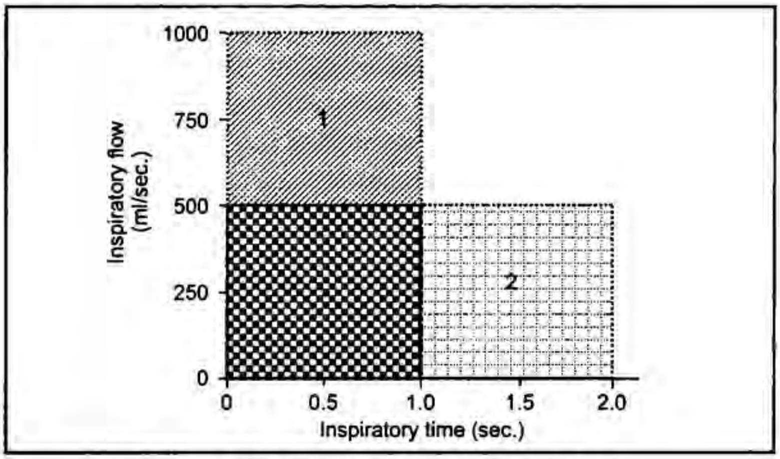


Figure 17.4: Factors influencing tidal volume

Mechanical inspiration is terminated after preselected inspiratory time.

Tidal volume = Inspiratory flow rate × Inspiratory time

The shaded curve represents a tidal volume of 500 ml. Tidal volume can be increased by increasing flow rate (as shown in 1) or by increasing inspiratory time (as shown in 2) or by increasing both.

TERMINOLOGY OF TIDAL VOLUMES

1. V_{Tdel} is the volume delivered from the ventilator.
2. V_{Teff} is the actual volume delivered to the patient.
3. V_{comp} is the compressible volume of the ventilator tubing.

GUIDELINES FOR SELECTION OF TIDAL VOLUME AND MINUTE VENTILATION

Traditional Inflation Volumes

In the earlier years during mechanical ventilation, traditional inflation volumes (10 ml -15 ml/kg) with mechanical sighs were used to prevent atelectasis. They not only resulted in a fall in cardiac output but also damaged the lung.

Disadvantages of Large Tidal Volume

These high inflation volumes are preferentially distributed to normal lung regions and leads to stress fractures in the walls of the alveoli and adjacent pulmonary capillaries, causing alveolar rupture with accumulation of alveolar gas in pulmonary parenchyma, mediastinum or pleural cavity. The pulmonary capillary damage causes a leaky capillary type of pulmonary edema. Lung damage is caused by a combination of overdistension and traction forces on lung units giving rise to parenchymal injury at the alveolar level, even in healthy lungs. Very large tidal volumes can cause respiratory alkalosis and decrease cardiac output by reducing venous return.

Lung Protective Inflation Volumes

The above observations have led to the restriction of the set V_{Teff} 6-8 ml/kg) or to the use of pressure-controlled ventilation in indicated patients, e.g. ARDS. The newer strategies are to use lower lung volumes with the use of PEEP to prevent the collapse of alveoli and small airways. The goal is to maintain an end inspiratory plateau pressure of less than 30 cm H_2O and permit permissive hypercapnia (less than 50 mm/Hg in normal patients).

Minute Ventilation

In a spontaneously breathing, healthy adult, the normal minute ventilation is 6 Lit/min (an average V_T of 500 ml multiplied by an average respiratory rate of 12 /min). The minute ventilation required to maintain acid base balance in intubated adult ICU patients of average size may range from 6 lit/min to 30 lit/min or more, varying with metabolic demands and the proportion of minute ventilation that constitutes dead space ventilation.

Minute ventilation is set according to the approximate ventilatory requirements of patients and to start with is set at 5-10 litre/minute. The decided minute ventilation is influenced by the nature of the disease and the altered respiratory mechanics, for which ventilatory support is offered.

With the guidance of arterial blood gas analysis, adjustment in the respiratory rate is done to alter delivered minute ventilation to match the patient's metabolic needs and to compensate for dead space ventilation.

PRACTICAL APPROACH TO DECIDE INFLATION VOLUME

The tidal volume should be set between 5-12 ml/kg body weight depending on respiratory mechanics. The selection of tidal volume in a given patient is influenced by the nature of the disease, the approximate minute ventilation requirement, pulmonary compliance, airway resistance, airway pressure, PaO_2 and $PaCO_2$. Therefore, modest V_T 8-10 ml/kg with 'physiological' (low level) CPAP/ PEEP 4-5 cm H_2O to prevent atelectasis is desirable for most patients.

Factors to be Considered

Desired Degree of Chest Expansion

Evaluate the desired degree of chest expansion by manual ventilation. Adequate tidal volume is the volume set on the ventilator giving the same degree of chest expansion.

Patient's Actual Body Weight

Usually the patient's actual body weight can be used for selecting the tidal volume unless the patient is significantly underweight or overweight. The initial tidal volume V_{Tdel} is usually set between 10-12 ml/kg of ideal body weight, with V_{Teff} between 8-10 ml/kg of ideal body weight.

Patients with Normal Lung Compliance

Patients with normal lung (e.g. in neuromuscular disease causing respiratory failure, CNS disease, coma, poisoning, postopertaive patient) who are first intubated and placed on ventilator, the initial settings for V_T and respiratory rate will provide only a rough estimate of the patient's required minute ventilation.

Patients with normal lung compliance may require a preset tidal volume of 12 to 15 ml/kg to produce a V_{Teff} of 8-10 ml/kg. An initial respiratory rate of 10 -12 / minute will provide minute ventilation more than sufficient to meet the ventilatory requirements of a normal subject.

Advantages of Low Tidal Volume

- To minimize the airway pressures
- To minimize the risk of barotrauma
- To allow permissive hypercapnia

Specific Conditions of Low Tidal Volume

Pulmonary Oedema and ARDS Patients

In patients whose lungs are stiff because of abnormal respiratory mechanics, tidal volume as low as 5- 6 ml/kg has been recommended in the early stages to prevent an increase in airway pressure requirement. Tidal volume can be set at 10 ml/kg and reduced to 5 - 6 ml/kg so that plateau pressure does not exceed 30 cm H_2O.

Severe Airway Obstruction or COPD Patients

Lower V_T may be selected in patients with severe airway obstruction where there is a concern about air trapping. COPD patients may also benefit from a reduced tidal volume setting or a higher flow rate.

- *Low tidal volume:*
  These patients have reduced expiratory flow rates due to decreased alveolar elastic recoil. Decreasing tidal volume by 100-200 ml in COPD patients reduces the expiratory time requirements and helps to prevent air trapping.
- *A higher flow rate:*
  A high flow rate is helpful to shorten the inspiratory time which helps to lengthen the expiratory time for COPD patients. Due to decreased alveolar elastic recoil, a longer expiratory time is needed for complete exhalation, because if there is not enough time for complete exhalation it will give rise to air trapping and V/Q mismatch which leads to hypoxemia and hypercapnia.

Patients with Lung Disease

Lower V_T may be selected in patients for whom the pressure required to deliver a V_T of 10-12 ml/kg is sufficiently high (>50 cm H_2O). Patients with lung disease may require a preset tidal volume of 14-15 ml/kg to produce an effective tidal volume of 8 to 10 ml/kg, because more volume will be lost in the ventilator circuit due to higher inflating pressures.

Patients with Lung Resection

Lower tidal volumes may also become necessary for patients with a reduction of lung volumes due to lung resection.

Hypovolaemic Patients

Hypovolaemic patients and those who are prone to develop barotrauma Because V_T is one of the important determinants of mean airway pressure, it should be restricted in hypovolemic patient, as well as in those who are prone to develop barotrauma.

Periodic breaths of 15-18 ml/kg (a 'sigh'-intermittent PEEP) to prevent microatelelectasis are used rarely and the sigh mechanism has disappeared from most new ventilators.

Disadvantages of Very Low Tidal Volume

- Progressive decrease in FRC leading to atelectasis
- Hypoventilation, hypoxemia
- Increase in venous admixture

Circuit Compressible Volume

While setting V_T it is important to appreciate the role of compliance of connector tubing and ventilator circuits. Ventilator circuits are compliant and expand during a positive pressure breath. This volume "lost" in the ventilator circuit is called the circuit compressible volume.

The volume lost is a function of the peak inflation pressure and the inherent compliance of the tubing, i.e. elasticity of the ventilator tubing. The set volume is almost never delivered to the lungs. The amount of circuit expansion results in a volume that does not reach the patient but is recorded as part of the expired tidal volume. Therefore, corrections of V_T are required, guided by the end-expiratory CO_2 and the $PaCO_2$. The actual volume delivered to the patient, V_{Teff} is the volume that can be calculated by the following equation.

Corrected V_T = Expired V_T – Circuit compressible volume

$$V_{Teff} = V_{Tdel} - V_{comp}$$

The higher the compliance of the ventilator tubings, the larger the difference between set and V_{Teff}. The compressible volume of the ventilator is more significant if small breathing volumes are used and therefore must be calculated more accurately. The usual compliance of connector tubing of adults is 3ml/cmH_2O, which means that 3 ml of volume is lost for every 1 cm H_2O increase in inflation pressure. Thus, inflation volume from the ventilator is 500 ml and the peak inspiratory pressure is 30 cm H_2O, then (3 × 30) 90 ml will be lost to expansion of the ventilator tubing and the inflation volume reaching the patient will be (500 – 90) 410 ml.

CHAPTER

18

Inspiratory Flow Rate

When initiating volume cycled ventilation the clinician must select an inspiratory flow rate, measured in litres per minute that determines how quickly the V_T is delivered. For a given pressure the tidal volume is mainly determined by the inspiratory flow rate.

Inspiratory flow rate is the most important determinant during the mechanical ventilation for attaining:

- Desired level of peak inspiratory pressure
  Flow rate is an important determinant, to deliver the needed PIP, and directly proportional to the inflation pressure (Fig. 18.1).
- Inspiratory time and I/E ratio (Fig. 18.1)

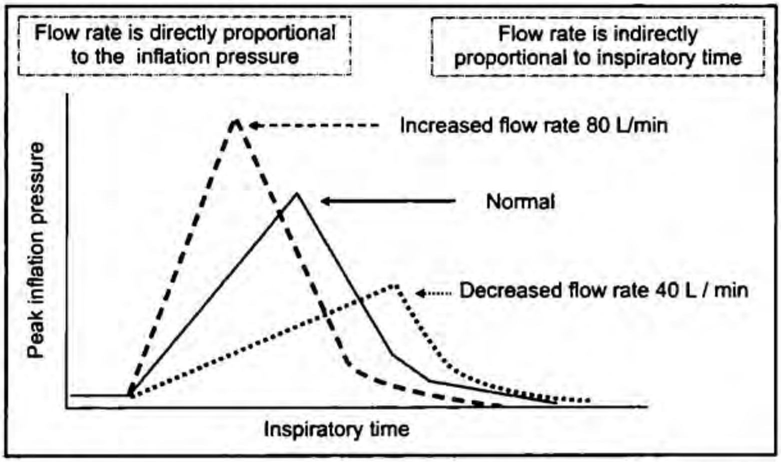


Figure 18.1: Relationship between flow rate, inflation pressure and inspiratory time

The inspiratory flow pattern also has an impact on inspiratory time and the I/E ratio. Flow rate is inversely proportional to the inspiratory time.

The slower the flow rate, the lower the inflation pressure and the longer the inspiratory time. The faster the flow rate, the quicker and higher the rise of inflation pressure and the shorter the inspiratory time.

INSPIRATORY FLOW RATE

The inspiratory flow rate influences the following:

- The distribution of inspired gas within lungs and therefore the distribution of ventilation within lungs
- The volume of CO_2 removed from the lungs
- Indirectly the V_D / V_T and Q_S / Q_T ratios, and the PaO_2

TYPES OF FLOW PATTERNS (FIG. 18.2)

- Square (constant) flow pattern
- Accelerating (ascending) flow pattern
- Decelerating (descending) flow pattern
- Sinusoidal or Sine wave flow pattern

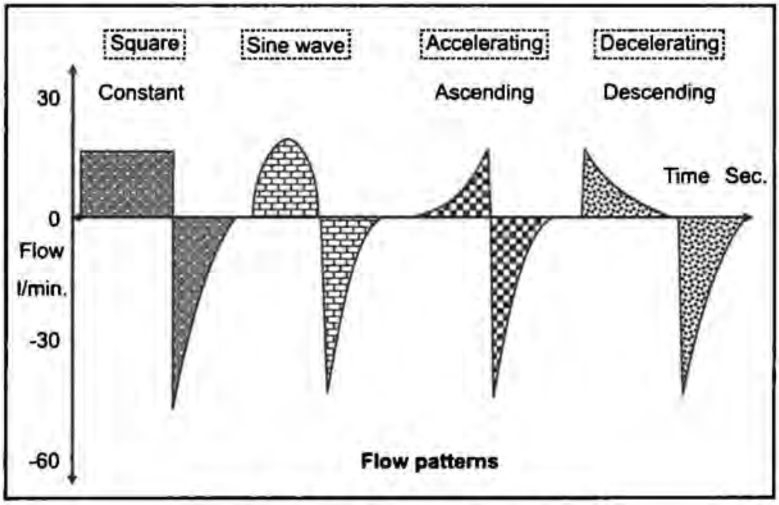


Figure 18.2: Types of flow patterns

Square or Constant Flow Pattern (Fig. 18.3)

Square or constant flow pattern is noted in volume controlled ventilation. The initial peak flow at the beginning of the inspiratory phase should help to overcome the airway resistance and parenchymal elastance. The remaining peak flow through the inspiratory phase should enhance gas distribution in the lungs.

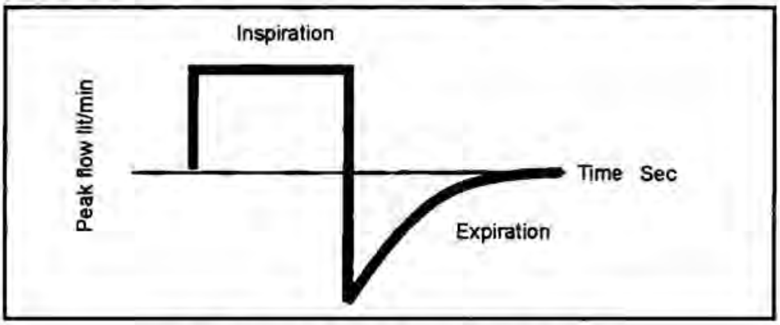


Figure 18.3: Square or constant flow pattern

Uses

Setting up the ventilator: Square flow pattern provides an even, constant peak flow during the entire inspiratory phase. The square flow pattern may be used upon setting up the ventilator.

To identify air trapping: The easiest way to identify air trapping is the flow waveform of constant flow ventilation (Fig. 18.4).

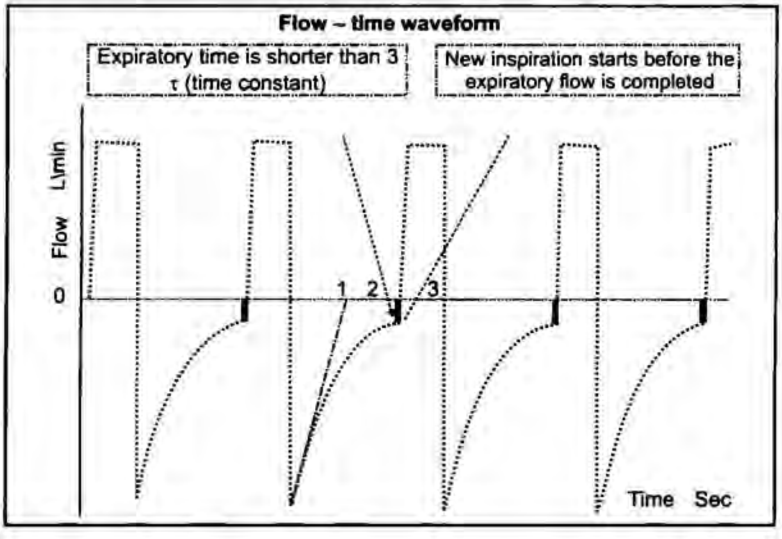


Figure 18.4: Auto-PEEP (For colour version *see* Plate 8)

This can be easily identified if new inspiration starts before the expiratory flow is completed. To perform the calculations only the mean inspiratory flow is used. Since the square wave flow pattern is only the flow pattern, in which the peak flow equals the mean inspiratory flow, the ventilator should be switched to a constant flow pattern prior to measurement of intrinsic PEEP.

Decelerating or Descending Flow Pattern (Fig. 18.5)

A decelerating flow pattern is noted in pressure-controlled ventilation. The decelerating flow pattern typically produces high initial inspiratory pressures which allow the set pressure to be reached early during the breath.

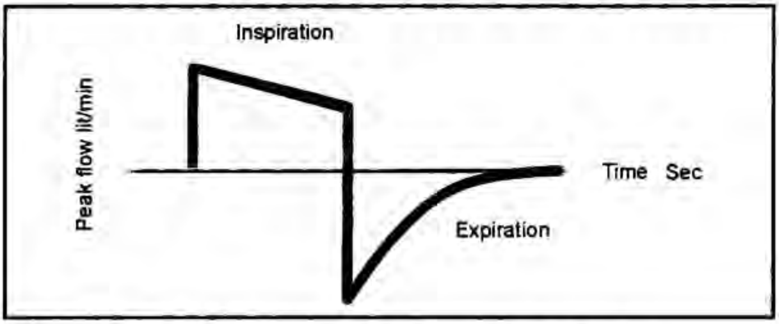


Figure 18.5: Decelerating or descending flow pattern

The decrease in flow may help pressure to remain constant throughout the inspiratory time and may improve distribution of ventilation, tidal volume and gas exchange. The decelerating inspiratory flow pattern reduces peak airway pressures.

Accelerating or Ascending Flow Pattern (Fig. 18.6)

Accelerating flow pattern increases flow in the remaining part of inspiratory cycle.

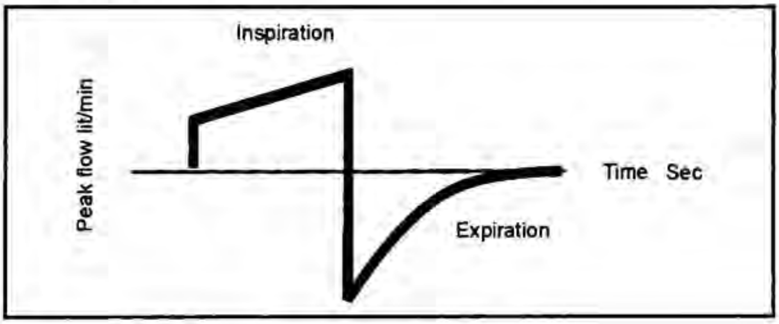


Figure 18.6: Accelerating or ascending flow pattern

Uses

With its increasing flow, the accelerating waveform may improve the distribution of ventilation in patients with partial airway obstruction.

Sinusoidal or Sine Wave (Fig. 18.7)

The sine wave is considered a more physiologic flow pattern.

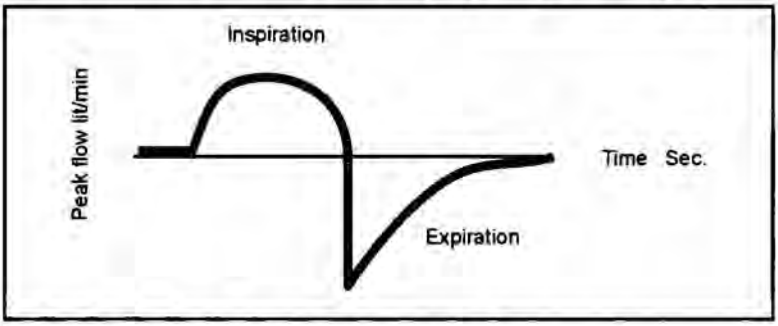


Figure 18.7: Sinusoidal or sine wave

The sine wave may also improve the distribution of ventilation and therefore improve gas exchange.

Comparisons of duration of inspiratory time for different inspiratory flow pattern using volume cycled ventilation—

- Inspiratory Time (Fig. 18.8):
  Note that the inspiratory time is longer with the decelerating and sinusoidal flow patterns than with constant flow, even though the

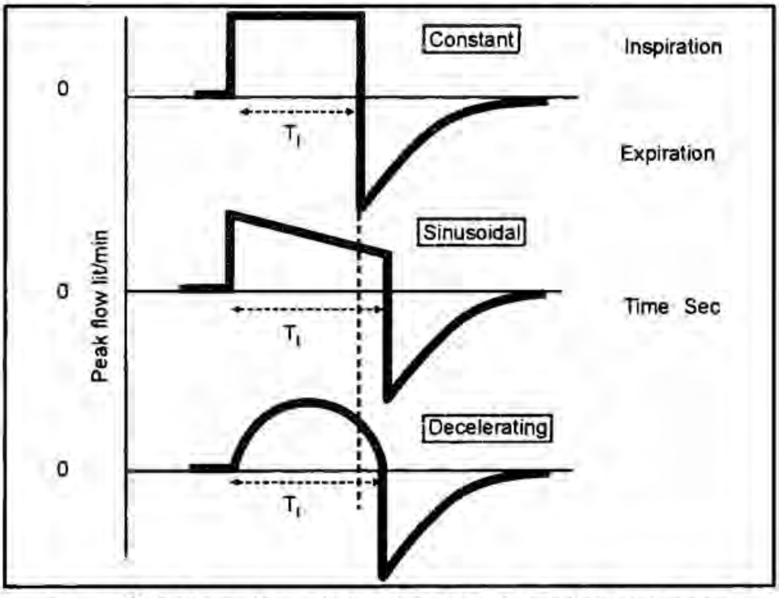


Figure 18.8: Comparisons of duration of inspiratory time for different inspiratory flow pattern (For colour version *see* Plate 8)

delivered volumes and peak inspiratory flow rates are equal. It means that the expiratory time with the decelerating and sinusoidal flow patterns will be shorter than with the constant flow pattern.

- Peak Inspiratory Flow Rate
  The constant flow pattern is the only flow pattern in which the peak flow rate equals the mean (average) flow rate. In all other flow patterns the mean flow rate is lower than the peak flow. Therefore, in decelerating, accelerating and sinusoidal flow pattern inspiratory time is longer for the same setting of peak flow and tidal volume, compared to constant flow pattern.

Ventilator Pressure Changes with Variation of the Inspiratory Flow in Volume Controlled Ventilation (Fig. 18.9)

In the pressure-time diagram the airway pressure is plotted against time. The pressure is expressed in cm H_2O and the time in seconds. The inspiratory flow is a measure of the velocity at which tidal volume is administered. It is expressed in L/min.

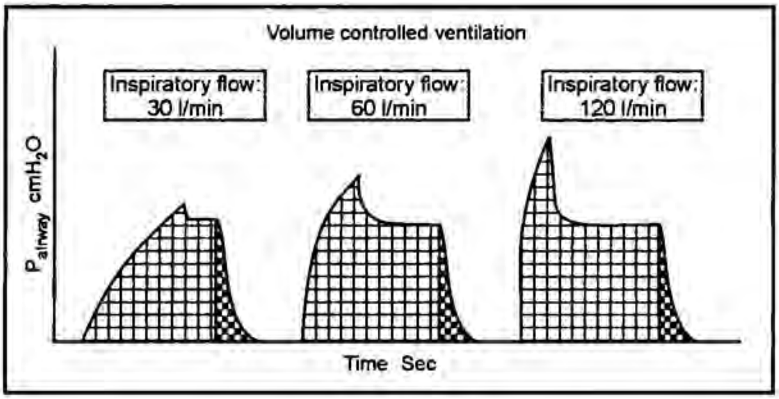


Figure 18.9: Pressure time diagram with variation of the inspiratory flow

At low inspiratory flow the pressure gradient is flat. A pressure peak just develops at a flow of about 30 L/min and I:E ratio of 1:2. The end-inspiratory plateau can just be seen. It would disappear completely if the inspiratory flow were even lower. With the increasing inspiratory flow the pressure gradient increases, simultaneously the peak inspiratory pressure is higher.

CONSIDERATIONS IN GUIDING FLOW RATE SELECTION

1. A minimum flow rate needed is—at least two and half or three times the minute volume.
2. Faster flows require greater central airway pressures to deliver the same V_T. Although airway resistance tends to disperse high airway pressures proximal to the alveolar level, some pathways may allow alveolar pressure to approach that of the airway opening. This rapid volume change at higher flow rate may increase shear forces, thereby risking further airway and parenchymal damage.
3. In certain patients, there are higher inspiratory flow demands. In these patients the respiratory centre signals that gas has to be delivered to the lung at a faster rate, particularly if the minute volume requirements are high. If the machine flow rate is set lower than patient's inspiratory flow demand, the patient's will pull or fight against the ventilator, thereby increasing energy consumption and contributing to anxiety.
4. Low inspiratory flow rate shortens the available expiratory time. In patient's with air flow obstruction, dangerous air trapping can occur if the flow rate is set too low.
5. Adjustments of the flow pattern may be made after stabilization of the patient.

Guidelines for Selecting Flow Rate in Various Clinical Condition

The inspiratory flow rate is usually set at 40-60 L/min in volume targeted ventilation. This can be increased up to 60-100 L/min in patients with high inspiratory demands. Better gas exchange and lower plateau pressure are demonstrated as inspiratory flow rate was increased from 40 -100 L/min in patient's with chronic airflow obstruction. The flow set at the ventilator must match the patent's inspiratory flow so that no negative pressure can occur.

$$\text{Flow rate} = \frac{\text{Tidal volume}}{\text{Inspiratory time}}$$

$$\text{Flow rate} = \text{Minute volume} \times \text{Sum of I:E ratio}$$

Flow Rate is Usually Set 10 L/min Higher to Meet Changing Minute Volume Requirements

Patients with High Ventilatory Requirement

Patient's with acute ventilatory failure, during weaning will be having high ventilatory requirement. This patient usually requires a peak flow rate at least four times the minute ventilation during constant flow pattern (e.g. flow setting of 60 Lit/minute is appropriate for patients with a minute ventilation of 15 litre/minute).

Patients who Initiate Breaths Spontaneously

In patients, who initiate breaths spontaneously, the flow rate should be adjusted at the bedside to match inspiratory effort. Because flow demands are highest in the beginning of inspiration, a decelerating flow profile often proves helpful. To switch to pressure-controlled ventilation for the same inspiratory tidal volume and inspiratory time, peak flow should be set 20-25% higher than in the constant flow mode.

Patients with Airflow Obstruction

Relatively higher flow rates should be used for patient's with airflow obstruction, to decrease inspiratory time.

ARDS Patient

Higher inspiratory flow rates would cause an increase in the peak inspiratory pressure. Hence, a lower flow rate can be used to decrease peak inspiratory pressures in these patients (as in ARDS).

Small Babies

A flow of 4-10 litre/min is normally sufficient for babies. If higher PIP is required, a correspondingly higher flow has to be used. The set flow determines the gradient of the pressure increase. With CPAP the flow rate in constant flow ventilator should be 2.5 to 3 times the minute volume, with a minimum of 3-4 L/min

Selection of Inspiratory Flow Rate and I:E Ratio Depending on Mode of Ventilation

In Volume Cycled Ventilation

- The clinician must select an inspiratory flow rate measured in L/min that determines how quickly the V_T is delivered. The time required to complete inspiration is a function of their mean (average) flow rate and tidal volume. The required time is called inspiratory time – T_I. The time the patient can exhale is determined by the inspiratory flow rate and frequency called expiratory time T_E.
- Flow patterns at constant flow rates, decelerating or sinusoidal flow patterns differ. The inspiratory time is longer for the same settings for peak flow for decelerating or sinusoidal flow patterns compared to constant flow.

- Peak flow rate is usually set in the constant flow mode about four time's minute volume. But during pressure controlled ventilation for the same inspiratory V_T and inspiratory time the peak flow rate should be set 20-25% higher due to the decelerating flow pattern.

In Pressure Cycled Ventilation

- The inspiratory flow rate has decelerating pattern to maintain constant pressure. Decelerating pattern is determined as a function of the driving pressure, the airway resistance and lung and chest wall compliance. The flow rate cannot be adjusted independently from these variables. Instead, the inspiratory time is adjusted by a timing mechanism that cycles the ventilator between inspiration and expiration. In most circumstances the I/E ratio is set at about 33%.
- Higher I/E ratio provides longer expiratory times. However, under conditions of high airway resistance, a short inspiratory time will result in a lower V_T, because the alveoli do not achieve their full equilibrium volume.
- If the I/E ratio is too low (>50% or 1:1) air trapping may result. I/E ratios greater than 1:1 are generally avoided except under special circumstances where it may be desirable.

Time Constants and Inspiratory Flow Rates (Fig. 18.10)

During mechanical ventilation, regional differences in resistance-compliance (time constants) within the lungs determine the distribution

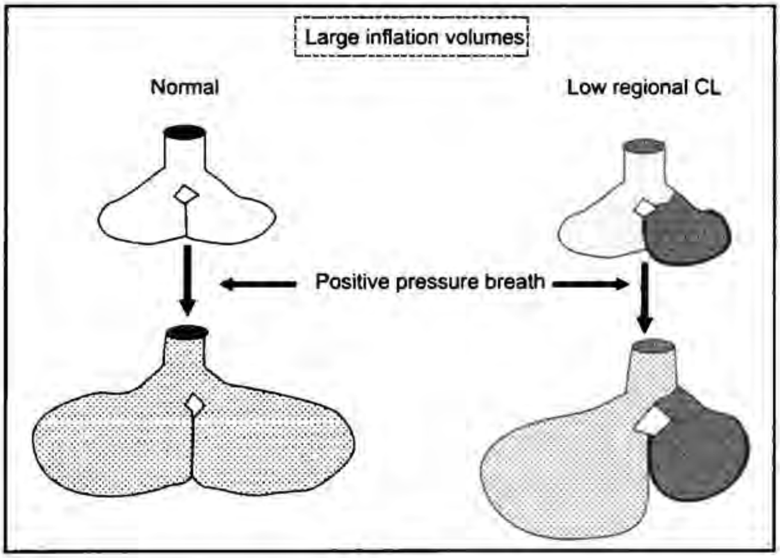


Figure 18.10: Lungs units with different time constants

of inspired gas and may seriously affect gas exchange. When two parallel lungs units with different time constants are ventilated, gas distribution between two units becomes more uneven with increasing flow rates.

At low flow rates airway resistance is a minor factor and distribution of inspired gas is more determined by regional differences in lung compliance. High inspiratory flow rates are needed if square wave forms is desired and also when the inspiratory time is to be shortened in order to maintain an adequate tidal volume.

Advantages of High Inspiratory Flow Rates

- High inspiratory flow rates have the ability to deliver a tidal volume in shorter time and thus maximize the time available for expiration.
- Carbon dioxide retention in the ventilator tubing will be prevented at high flow rates.

Disadvantages of High Inspiratory Flow Rates

A serious side effect of high flow rate is barotrauma. Maldistribution of ventilation results in a rapid pressure increase in the non-obstructed or non-atelectatic alveoli (normal alveoli) giving rise to increased incidence of alveolar trauma and rupture.

Advantages of Low Inspiratory Flow Rates

- Reduction in peak airway pressure producing less chance of barotrauma
- To minimize likelihood of coughing in diseased airway
- To decrease turbulence in diseased airways
- To improve distribution of ventilation from lung areas with low resistance (fast compartment) to areas with high resistance

Disadvantages of Low Inspiratory Flow Rates

Due to shortening of expiratory time, hypercapnia is a common feature and air trapping may occur in patients with airway obstruction.

CHAPTER

19

Peak Inspiratory Pressure

MEAN AIRWAY PRESSURE (MAWP) - P_{MEAN}

The average pressure within the airway during one complete respiratory cycle. It is an essential factor for oxygenation.

PEAK INSPIRATORY PRESSURE (PIP) OR PEAK AIRWAY PRESSURE (PAP)

The maximum pressure measured during one respiratory cycle, usually at the end of inspiration. Like tidal volume in volume-controlled

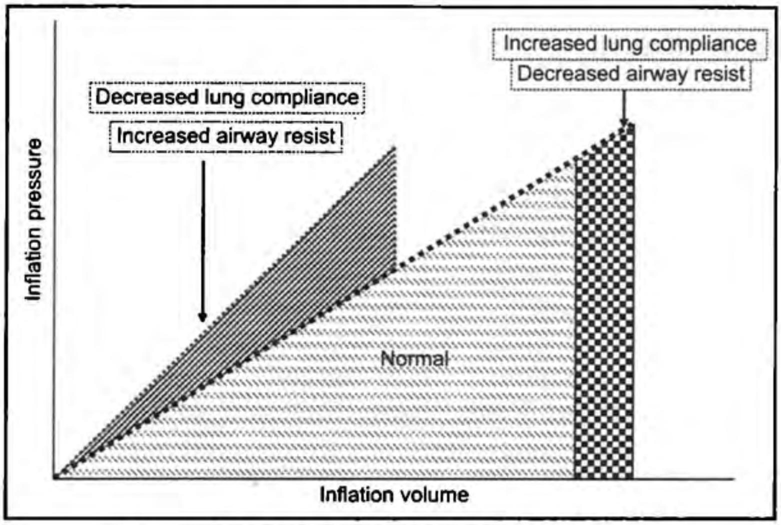


Figure 19.1: Determinants of inflation volume during pressure-controlled ventilation

ventilation, PIP is the main parameter to be set during pressure controlled/limited ventilator.

For this reason, the patient may receive a smaller volume when the preset pressure is reached prematurely as would happen under conditions of low compliance and high airway resistance or larger volume when compliance improves or resistance decreases (Fig. 19.1).

The appropriate setting of PIP depends on many factors—

- Estimated tidal volume requirement of a patient
  The starting level of PIP must be considered carefully. The PIP is preset according to the estimated tidal volume requirement of a patient.
- The lowest PIP that adequately ventilates is appropriate—
  The appropriate setting can be determined by determining the pressure to cause an adequate chest expansion while bagging and to open atelectatic alveoli in case of RDS.
- Evaluation of the patient—
  The starting level of PIP also depends on the type and severity of the disease, change in lung mechanics such as lung compliance and resistance.

Precautions

PIP of < 30 mmHg is usually sufficient for most respiratory disorders. High PIP (>30 mmHg) is avoided because of the risk of air leaks and barotrauma.

Factors Influencing Airway Pressure in Volume-controlled /Volume-limited Ventilation

In volume-controlled ventilation, the tidal volume is preset and the pressure is variable. The airway pressure depends on the mechanical characteristics of the lung (resistance and compliance), inspiratory flow and tidal volume (Fig. 19.2).

When using volume controlled ventilation, all airway pressure (including PAP and MAWP) are directly related to tidal volume V_T, airway resistance (R), peak inspiratory flow rate (PIFR) and inversely related compliance (C) (Fig. 19.3).

Relationship between Peak Airway Pressure and Compliance

Lungs with Normal Compliance

In lungs with normal compliance, about 50% of the airway pressure is transmitted to the thoracic cavity.

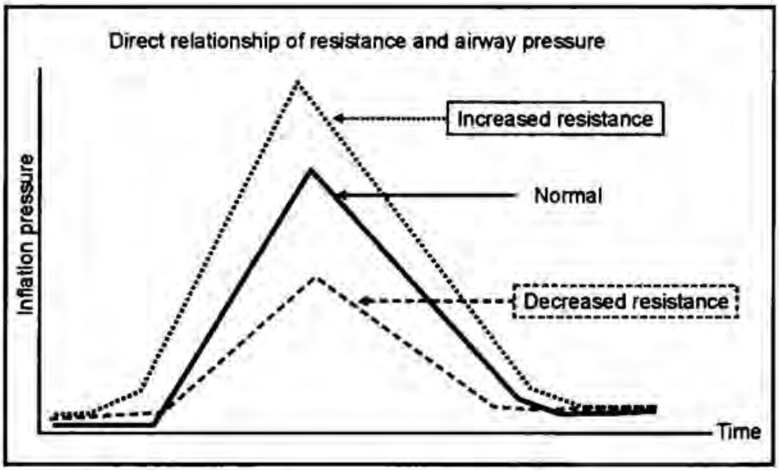


Figure 19.2: Direct relationship of resistance and airway pressure during volume-controlled ventilation

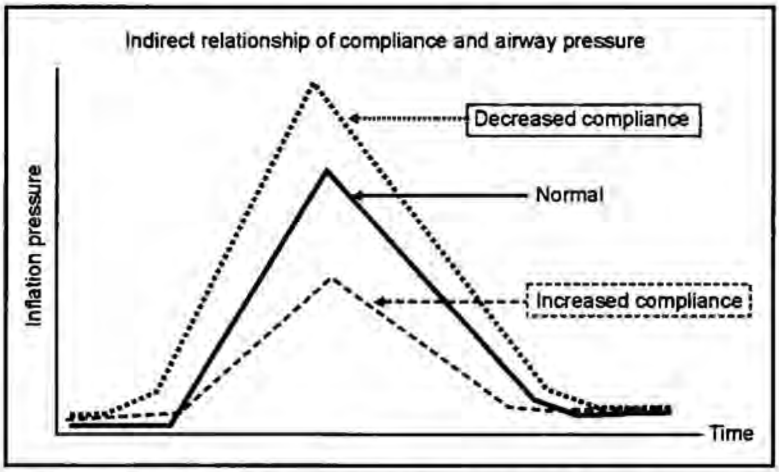


Figure 19.3: Indirect relationship of compliance and airway pressure during volume-controlled ventilation

Non-compliant or "Stiff" Lungs

High levels of PAP or PEEP may be required to ventilate and oxygenate patients with low compliance. However, in non-compliant or "stiff" lungs (atelectasis, ARDS), the pressure transmitted to the thoracic cavity is much less due to the dampening effect of the nonelastic lung tissues. The decrease in cardiac output due to excessive PAP or PEEP is less severe in these patients than if the same pressures are applied to lungs with normal or high compliance.

Clinical Uses of PIP

Improving Oxygenation

The lowest PIP necessary to ventilate the patient adequately is optimal in most cases. This will be associated with increased V_T, increased CO_2, elimination, decreasing $PaCO_2$. All these effects are due to the rise of mean airway pressure which improves oxygenation. PIP can affect PaO_2 and $PaCO_2$

To Reveal Changes in Lung Mechanics

- High-peak airway pressure that is associated with a normal plateau (end-inspiratory) pressure reflects abnormally increased resistance, which may be due to narrow tracheal tube or bronchospasm.
- A small difference between a high-peak airway pressure and plateau pressure reflects low lung compliance.

Complications of PAP

The main complication of PAP is barotrauma. Barotrauma is the term used to describe lung tissue injury or rupture which result from alveolar overdistension.

Factors Influencing Barotrauma

- *Ventilator parameter*
  - PIP > 50 cm H_2O
  - Plateau pressure >30 cm H_2O
  - PEEP >10 cm H_2O
- *Duration of IPPV*
- *Condition of the patient*
  COPD patients are more susceptible to barotrauma due to air trapping and weakened parenchymal areas (e.g. lung blebs and bullae).
- *Uneven distribution of ventilation*
  In patients with significant airway obstruction and lung parenchymal disease, a mechanical tidal volume tends to preferentially distribute to areas of low resistance and high compliance during the early portion of inspiration. This may result in transient elevated alveolar pressures with resultant overdistension and rupture despite what would normally be accepted as a "safe" pressure.

Precautions to Avoid Barotrauma

The best precaution to avoid barotrauma is to minimize PIP. If PIP is minimized there is decreased incidence of barotrauma with resultant air leaks (pneumothorax and pneumomediastinum).

Alarm Settings

Low-pressure Alarm

The disconnect alarm on most ventilators is based on airway pressure setting. The low-pressure alarm at 10-15 cm H_2O below peak inspiratory pressure will:

- Detect ventilatory disconnection
- Detect a leak
- Improved resistance or compliance

High-pressure Alarm

Similarly, setting the high-pressure alarm 5-10 cm H_2O above peak airway pressure will give

- Early warning of increased resistance
- Decreased compliance
- The patient coughing or 'fighting' the ventilator

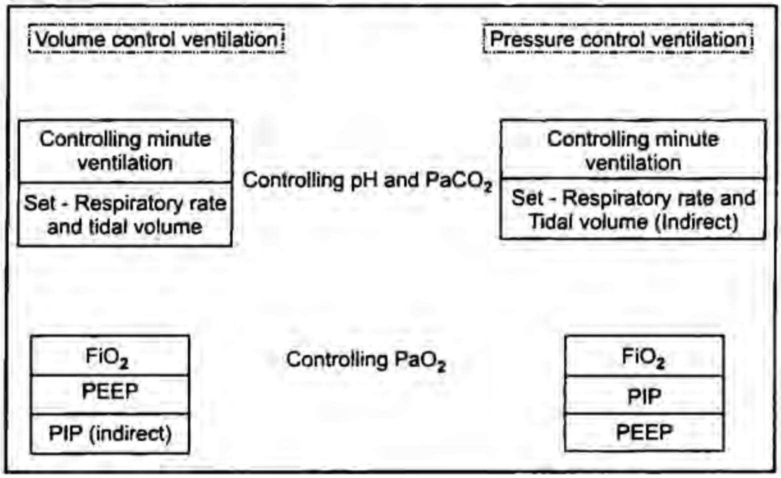
Degree of negative airway pressure swing *at the beginning of inspiration may give a clue:*

- To possible increase in ventilator resistance
- Increased work of breathing

Such increased inspiratory effort can be managed by—

- Increasing the ventilator's sensitivity
- Switching to a decelerating flow patterns to minimize the pressure rise time
- Ruling out sources of increased inspiratory resistance (e.g. occluded tracheal tube).

Summary: Settings to improve oxygenation and ventilation in controlled ventilation


Volume control ventilation
Pressure control ventilation
Controlling minute ventilation
Set - Respiratory rate and tidal volume
Controlling pH and $PaCO_2$
Controlling minute ventilation
Set - Respiratory rate and Tidal volume (Indirect)
FiO_2
PEEP
PIP (indirect)
Controlling PaO_2
FiO_2
PIP
PEEP

CHAPTER

20

Breathing Cycle and Inspiratory/ Expiratory Time

RESPIRATORY PATTERN

A respiratory pattern describes the course of a single breathing cycle.

Breathing Cycle or Respiratory Cycle - T_{Tot}

A breathing cycle or respiratory cycle is the time from the beginning of inspiration to the end of expiration. The total time of each respiratory cycle or breath (T_{Tot}) consists of the inspiratory time (T_I) and the expiratory time (T_E). The following equation applies:

$$T_{Tot} = T_I + T_E$$

Respiratory Time Ratio or I:E Ratio

The ratio of inspiration time to the expiration time is referred to as the respiratory time ratio or inspiration time to the expiration time ratio (I: E ratio)

$$\text{Respiratory time ratio} = T_I : T_E$$

If inspiration takes 2 seconds, expiration 4 seconds, the breathing cycle time $T_{Tot} = 6$ sec, then the respiratory rate = 10/min and the I:E = 1:2

Respiratory Rate – RR or f

The respiratory rate is the number of breaths per minute.

$$\text{RR or } f = 60/T_{Tot}$$

Respiratory Minute Volume (RMV)

The respiratory rate is multiplied with the tidal volume (V_T) the result is the respiratory minute volume (RMV)

$$\text{RMV} = V_T \times \text{RR}$$

Inspiratory Flow Rate and I/E Ratio

Inspiratory flow rate measured in l/minute that determines how quickly the V_T is delivered.

Inspiratory Time – T_I

The time required to complete inspiration is a function of the inspiratory flow rate and tidal volume settings.

$$T_I = V_T/\text{Average flow rate}$$

Thus, the I/E ratio is also affected by the inspiratory flow rate.

Ventilatory Parameters for Adequate Respiratory Support

In order to provide adequate respiratory support ventilatory parameters must be chosen that

1. Provide adequate tidal volume
2. Time for even gas distribution – Time for inspiration
3. Time for expiration.

Selection of Duration of Inspiratory Time

1. Inspiratory time must be selected to allow sufficient time for all lung segments to be inflated.
2. If inspiration starts before the lung has completely emptied, this will result in air trapping and inadvertent positive end-expiratory pressure.
3. Inspiratory time can be set either as a percentage of the total respiratory cycle or as a fixed time in seconds depending on the ventilator.
4. During intermittent positive-pressure ventilation in a normal subject the duration of inspiration is usually set at 1-1.5 seconds.

Physiological Effects of Prolonging Inspiratory Time

Prolonging inspiratory time, generally by adding a pause and often used in combination with rapid decelerating flow (pressure targeted breath), also increases mean inflation pressure and lengthens gas mixing time in the lung. Moreover, if the resultant expiratory time is inadequate for the lung to return to its relaxed volume (i.e. FRC), intrinsic PEEP (air trapping) develops.

Relationship of Peak Alveolar Pressure to MAP Using Various Strategies to Increase Mean Alveolar Pressure (Fgi. 20.1)

With increase in extrinsic PEEP, the relationship is linear. If increase in inspiratory time is not associated with intrinsic PEEP, mean airway pressure is increased without increase in peak alveolar pressure. However, when air trapping and intrinsic PEEP develop, either a higher peak alveolar pressure is needed for constant tidal volume (solid line) or tidal volume decreases for a constant peak alveolar pressure (dotted line).

- Increased duration of inspiration will increase gas mixing time and may improve V/Q matching in infiltrative lung disease.
- Improvement in gas exchange associated with long inspiratory time strategies may be merely a PEEP phenomenon.

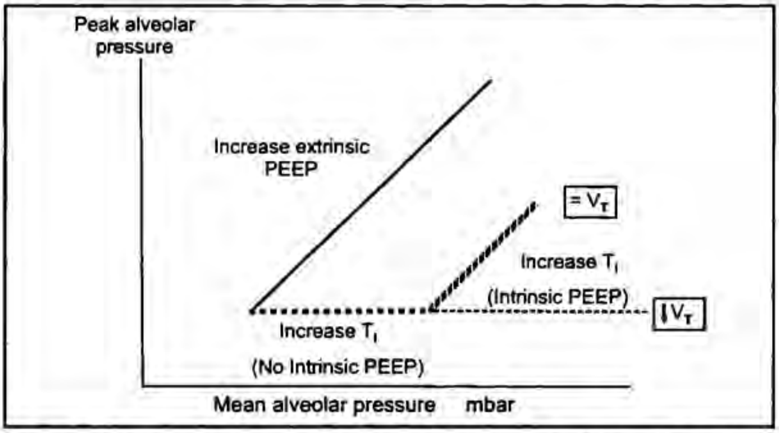


Figure 20.1: Relationship of peak alveolar pressure to MAP using various strategies to increase mean alveolar pressure (For colour version *see* Plate 9)

- A long inspiratory time increases mean intrathoracic pressure reducing venous return and cardiac output.

Effects of Long Inspiratory Times on Airway Pressure and Alveolar Volume

1. The inspiratory and expiratory ratios are such that alveoli are at baseline volume for three fourths of the time (Fig. 20.2).
2. Inspiratory time has been extended so that alveoli are at inspiratory volume for a longer fraction of time, yet expiratory time is adequate for return to the baseline volume. Under these circumstances, mean alveolar pressure is increased, but peak and baseline alveolar pressures are not affected (Fig. 20.3).
3. Inspiratory time has been extended to the point that expiratory time is inadequate for a return to baseline volume. Under these circumstances, mean alveolar pressure has increased further. However, baseline alveolar pressure also has increased further giving rise to "intrinsic" PEEP.

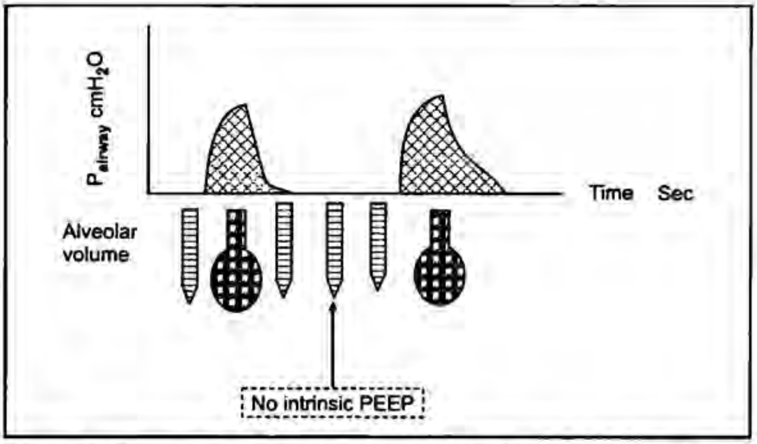


Figure 20.2: Alveoli are at baseline volume for three fourths of the time

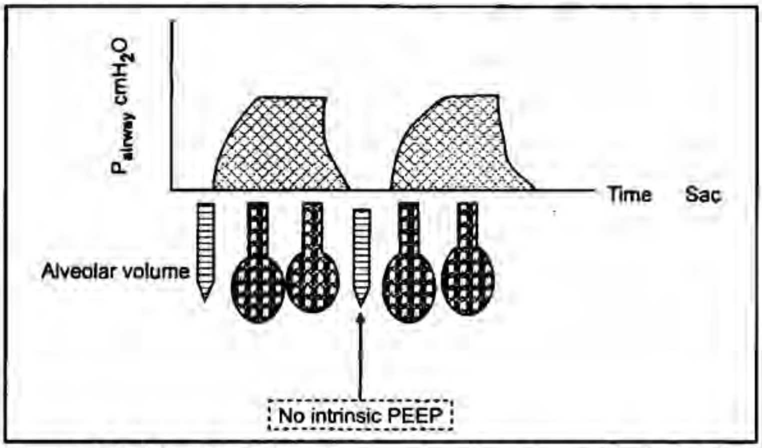


Figure 20.3: Alveoli are at inspiratory volume for a longer fraction of time

- Intrinsic PEEP reduces tidal volume in pressure controlled breath (Fig. 20.4).

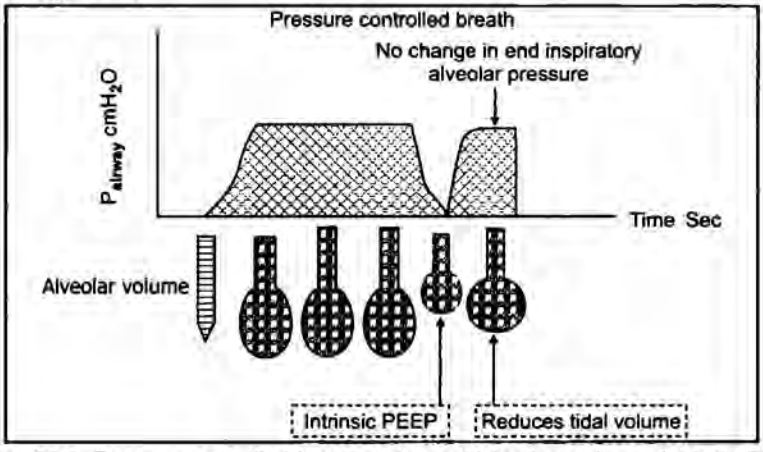


Figure 20.4: Pressure controlled breath reduces tidal volume due to intrinsic PEEP

- Intrinsic PEEP increases end inspiratory alveolar pressure in volume (Fig. 20.5).

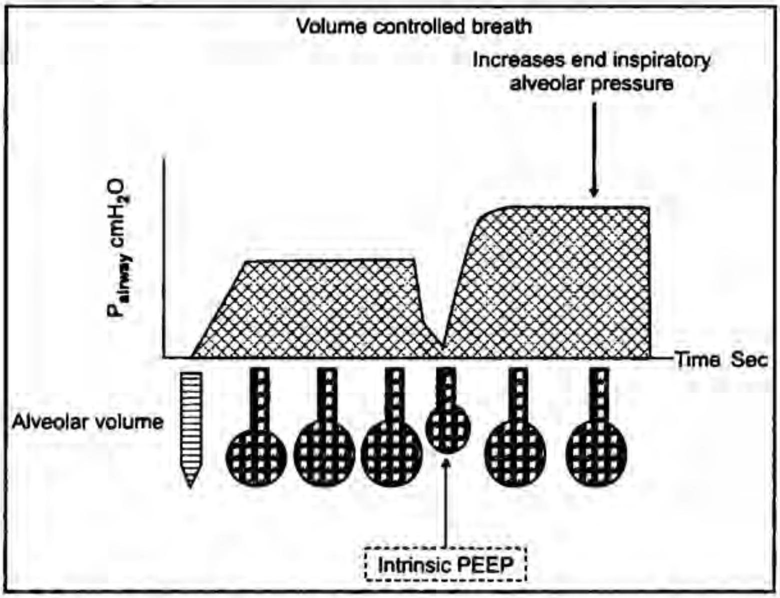


Figure 20.5: Volume controlled breath increases end inspiratory alveolar pressure due to intrinsic PEEP

Physiological Effects of Short Inspiratory Time

- A short inspiratory time causes poor distribution of the inspired gas due to the characteristics of 'fast' and 'slow' alveoli.
- A shortening of the time constant occurs when compliance or resistance or both are low

Inspiratory Time	*Venous Return Cardiac Output*	*Distribution of Inspired gases*
More than 1.5 sec.	Bad	Good
Less than 1 sec.	Good	Bad

Selection of Duration of Inspiratory Time in Different Diseases

Heterogeneous Lung Disease

In heterogeneous lung disease with varying regional time constants, a short inspiratory time may not be sufficient to inflate all lung segments, but may contribute to underventilation and underinflation. Similarly, sufficient expiratory time must be provided for all lung segments to empty.

ARDS Patient

An ARDS patient has a decreased compliance (stiff lungs) and normal resistance. In this patient the time constant and corresponding time for pressure equilibrium will be shorter. Short inspiratory and expiratory times may be appropriate during the period of peak severity of the disease, but insufficient after recovery from ARDS, when compliance is higher.

Infants with Disease that Causes Increased Airway Resistance

Infants with disease that causes increased airway resistance, such as Meconium aspiration syndrome (MAS), Asthma or Bronchopulmonary dysplasia (BPD) will have increased time constants due to slower filling and emptying of the alveoli. In disorders with increased airway resistance like MAS use shorter inspiratory time. In infants with bronchiolitis and in children with asthma, the expiratory time may have to be lengthened to avoid PEEP. Once set, inspiratory time is usually not changed unless there is persistent hypoxemia unresponsive to changes in PIP & FiO_2. In low compliance condition like RDS use closer to 0.5 seconds

EXPIRATION TIME - Te

The inspiratory flow rate and frequency (respiratory rate) determines the time during which the patient can exhale.

Clinical Importance of Sufficient Expiratory Time

1. To allow for complete deflation of the lungs
2. To allow for the lowest effective mean airway pressure
3. To compensate for the adverse effects on circulatory hemodynamics.

Inspiratory and Expiratory Ratio

Conventional ventilatory techniques employ I/E ratios of <1:1 and more typically <1:2. The interval during which gas actually enters the lung normally should not be <1.5 seconds or an unacceptably high peak airway pressure may result. Decreasing the I/E ratio will increase mean airway pressure and may cause air-trapping and generate intrinsic PEEP.

SELECTION OF I:E RATIO

- It is usually kept in the range between 1:2 to 1:4
- I/E ratio should be greater than 1:1.5 to allow for the lowest effective mean airway pressure,
- A longer I:E ratio may be used in patients needing additional time for exhalation because of the possibility of air trapping and auto-PEEP

Inverse I:E ratio

- Inverse I:E ratio has its inherent cardiovascular complications, so it should not be used as the initial setting.
- Inverse I:E ratio should be tried only after traditional strategies have failed to improve a patient's ventilation and oxygenation status.
- Inverse I:E ratio has been used to correct refractory hypoxemia in ARDS patients with very low compliance.

Manipulations of Control of I:E Ratio

Inspiratory time settings determine the I/E ratio. Thus, if tidal volume and inspiratory flow rates are constant, increasing the respiratory rate reduces the expiration time and thereby decreases the I/E ratio. Conversely, a decrease in respiratory rate under similar circumstances will increase the I/E ratio.

Depending on the features available on the ventilator, the I:E ratio may be altered by manipulating any one or a combination of the following controls.

- Inspiratory time
- Flow rate
- Inspiratory time %
- Respiratory rate
- Minute volume (tidal volume and respiratory rate)

I Time % and I:E Ratio

Some ventilators permit the I:E ratio to be preset, usually by setting an I time % (percent inspiratory time). In these ventilators, the flow rate is automatically adjusted by the ventilator to maintain a constant I:E ratio regardless of changes in tidal volume or respiratory rate. Pressure-controlled inverse ratio ventilation is a ventilatory mode that specifically uses this feature to achieve better alveolar recruitment during ventilation though cardiovascular side effects are significant with airway trapping.

I Time % and I:E Ratio Equivalent

It is generally desirable to maintain I/E ratio between 1:1.5 and 1:3. The tidal volume settings thus limit the range of acceptable inspiratory flow rates.

I Time %	*I : E ratio*
14.3%	1:6
16.7%	1:5
20%	1:4
25%	1:3
33.3%	**1:2**
50%	1:1
60%	1.5:1
60.7%	2:1

Effects of Flow Rate on I Time and I:E Ratio when the V_T and RR Kept Unchanged

I time and I:E ratio are inversely related. A higher I time leads to a lower I:E ratio. Adjusting the flow rate is the most common method to change an I:E ratio because the flow rate control is a feature available on almost all ventilators.

Parameter change	*I time*	*E time*	*I:E ratio*
Increase flow rate	Decrease	Increase	Increase
Decrease flow rate	Increase	Decrease	Decrease

Effects of V_T on I:E ratio

Parameter change	*I time*	*E time*	*I:E ratio*
Increase V_T	Increase	Decrease	Decrease
Decrease V_T	Decrease	Increase	Increase

Effects of RR on I:E ratio

Parameter change	*I time*	*E time*	*I:E ratio*
Increase RR	Minimal change	Decrease	Decrease
Decrease RR	Minimal change	Increase	Increase

Inspiratory Plateau or Inspiratory Pause Time (Fig. 20.6)

Physicians can maintain a patient's lung inflation at full inspiration by setting an inspiratory pause or plateau. This gives more even distribution of inspired gas. Inspiratory pause time is an important component of mean airway or more precisely of mean alveolar pressure. This is seen with volume-controlled ventilation with high flow.

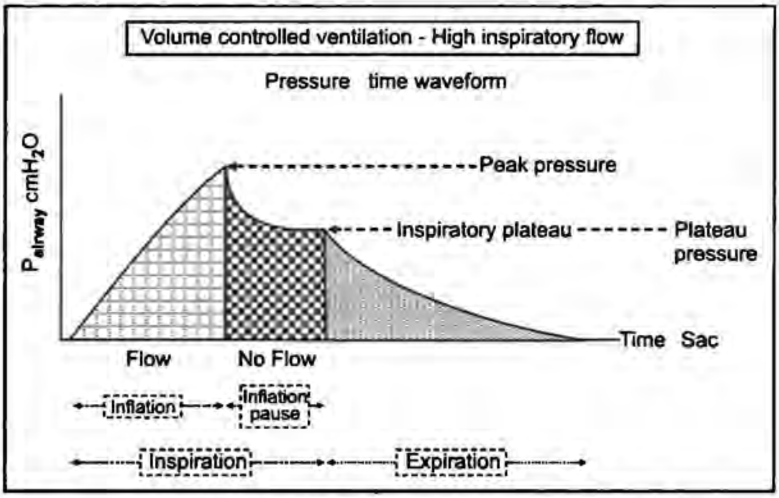


Figure 20. 6: Inspiratory plateau

The mean alveolar pressure relates intimately to efficiency of CO_2 removal as well as oxygenation. An inspiratory pause shortens the expiratory phase of the respiratory cycle, decreasing the I/E ratio.

Disadvantage of Inspiratory Plateau or Inspiratory Pause Time
Inspiratory plateau increases mean alveolar pressure and increases intrathoracic pressure for longer duration, decreasing venous return.

Usefulness of Inspiratory Pause

- *Inspiratory pause can decrease I:E ratio:*
  The inspiratory pause control occludes the expiratory port (preventing exhalation) for a set time following the delivery of the V_T. During volume cycled ventilation the ratio can vary independently of the flow rate by setting inspiratory pause.
- *Estimation of static compliance (Fig. 20.7):*
  The physician can estimate alveolar pressure at end-inflation by setting an inspiratory pause during one respiratory cycle in volume controlled ventilation. With flow in the airway stopped, alveolar pressure equilibrates with that measured at the proximal airway by the ventilator manometer.

  Any changes in transairway or transthoracic pressure help to monitor lung mechanics and help to reduce stay in ICU.

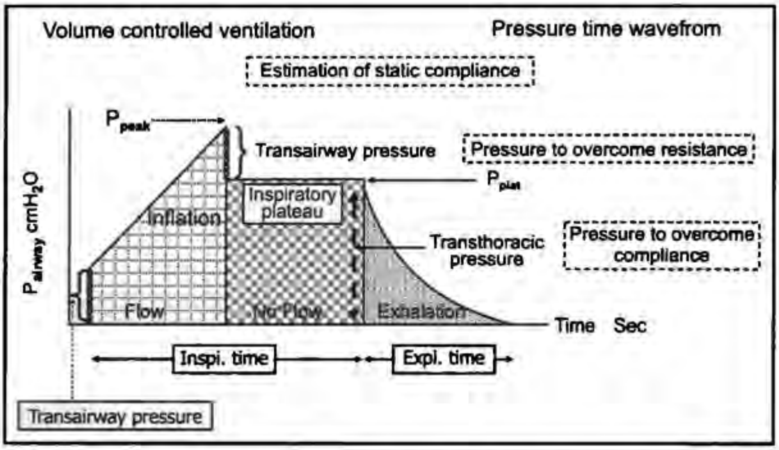


Figure 20.7: Volume-controlled ventilation (For colour version *see* Plate 9)

- *Implement IRV:*
  In volume-cycled ventilation it is easy to implement IRV with the use of end-inspiratory pause. But it can be implemented in well sedated, passive patients.

CHAPTER

21

Distribution of Inspired Gas and Ventilator Rate

DISTRIBUTION OF INSPIRED GAS

Dead Space Ventilation or Wasted Ventilation

Dead space ventilation is expressed as the product of dead space volume and the respiratory rate. Dead space ventilation therefore increases linearly with breathing frequency.

$$\dot{V}_D = \text{(Dead space volume) } V_D \times RR$$

Alveolar Ventilation or Effective Ventilation

Alveolar ventilation is defined as that proportion of the tidal or minute volume, which takes part in gas exchange.

$$\dot{V}_A = (V_T - V_{D\ phys}) \times RR$$

The normal value for alveolar ventilation is about 3.5 to 4.5 lit/ minute in adults.

Its importance lies in the fact that it is the pulmonary factor which controls the excretion of carbon dioxide by the lungs. Alveolar ventilation (excretion of carbon dioxide by the lungs) directly related to the tidal volume, physiological dead space and respiratory rate.

As the respiratory rate increases and tidal volume decreases, dead space ventilation increases at the expense of alveolar ventilation. Increasing tidal volume can compensate increased dead space ventilation. If tidal volume is not increased, then increased respiratory rate will lead to an increase in the work of breathing.

Alveolar air equation

$$\text{Alveolar oxygen tension} = \text{Inspired oxygen tension} - \frac{\text{Alveolar carbon dioxide tension}}{\text{Respiratory quotient}}$$

$$PAO_2 = PIO_2 - \frac{PACO_2}{RQ}$$

Thus, if the alveolar carbon dioxide tension ($PACO_2$) rises and the alveolar oxygen tension (PAO_2) falls the result will be arterial hypoxemia. The most obvious response to hypoxia is hyperventilation due to peripheral chemoreceptor stimulation. Hyperventilation leads to a fall in $PACO_2$ and a fall in the $PACO_2$ will allow the alveolar oxygen tension (PAO_2) to rise. A higher alveolar oxygen tension will increase the saturation of pulmonary capillary blood.

GOALS OF SELECTING VENTILATORY RATE

1. To Provide Eucapneic Ventilation
   The initial respiratory (frequency) is the number of breaths per minute that is intended to provide eucapneic ventilation ($PaCO_2$ at patient's normal).
2. To Allow Permissive Hypercapnia
   Permissive hypercapnia is usually achieved by reducing the V_T rather than the respiratory rate. Adjust the rate according to the need after ABG but it is necessary to keep airway pressure <35 cm H_2O and $PaCO_2$ 40-50 mm Hg.

SELECTION OF VENTILATORY RATE

Methods of selecting the initial respiratory rate assumes that both CO_2 production and physiological dead space are normal.

Factors Influencing Selection of Ventilator Rate Depends on the

1. *The Initial Ventilator Rate According to Age of the Patient*
   - Newborn infant-25-30/minute
   - 1 yr old - 20-25/minute
   - Adolescent - 15-20 / minute

   In adults it is usually between 10 to 12 breaths per minute. This rate, coupled with 10-15 ml/kg tidal volume, usually produces a minute volume that is sufficient to normalize the patient's $PaCO_2$.

2. *Estimate the Patient's Minute Volume and Tidal Volume Requirement*

   The respiratory rate together with tidal volume is the primary determinant of minute ventilation and hence CO_2 elimination. An alternative method of selecting the initial respiratory rate is to estimate the patient's minute volume requirement and divide the estimated minute volume by the tidal volume.

$$\text{Respiratory rate} = \frac{\text{Estimated minute volume}}{\text{Tidal volume}}$$

Since $MV = V_T \times RR$, increasing RR lowers the $PaCO_2$ level.

In a normal healthy adult the normal minute ventilation is 6 lit/min. An average tidal volume is 500 ml multiplied by an average respiratory rate of 12/min. In ICU settings the minute ventilation required to maintain ABG may be between 6 lit/min to 30 lit/min depending on ventilatory requirement.

3. *Adjustment Needed According to the Needed $PaCO_2$*

It is clear that if RR is increased then it lowers the $PaCO_2$ level. 10 –12 breaths/min are needed to maintain ABG within the normal limit assuming that both CO_2 production and physiological dead space are normal. The respiratory rate influences expiratory time. Inspiratory flow rate and tidal volume influences the inspiratory time and indirectly both influences expiratory time. The most important thing to remember is that the respiratory rate is the primary control to alter the $PACO_2$

EVALUATION OF THE PATIENT

1. *Check blood gases within 15-30 minutes*
   After placing the patient on a ventilator, blood gases should be obtained within 15-30 minutes after the patient has been stabilized to assess both ventilation and oxygenation.
2. *Increase frequency if the $PaCO_2$ is too high*
   Increase frequency of the patient who is having higher $PaCO_2$ than normal. Since the $PaCO_2$ varies inversely with the alveolar minute ventilation, a higher than normal $PaCO_2$ (e. g.>45 mm Hg in normal patient or > 50 mm Hg for patients with chronic CO_2 retention) means the patient's minute volume should be increased, usually by increasing the respiratory rate.
3. *Decrease frequency if the $PaCO_2$ is too low*
   In patients with lower than normal $PaCO_2$ (e.g. < 35 mm Hg in normal patient or < 40 mm Hg for patients with CO_2 retention) indicates that the minute volume should be decreased, usually by decreasing the respiratory rate.

Precautions

Since increasing the tidal volume results in higher airway pressure on a volume-limited ventilator, it is usually more appropriate to increase the minute volume by raising the respiratory rate. It is important to realize that reducing the respiratory rate in time-cycled ventilator should be usually accompanied with readjustment of the I/E ratio to prevent long inspiratory times.

Disadvantages of Using Higher Respiratory Rate

- Higher rates allow less time for expiration
- May cause air trapping, especially in the presence of increased airway resistance.

Clinical Conditions Using Higher Rate

- *CO_2 production is elevated*
  During laparoscopic surgery or in conditions of increase in metabolic rate, CO_2 production is elevated. In these patients higher rate is needed.
- *Infants with pulmonary hypertension and right to left shunting*
  In this patient it is important to reduce $PaCO_2$ which will increase pH. This helps to reduce pulmonary arterial resistance and also shunting associated with increased $PaCO_2$.
- *MAS - where CO_2 retention is a major problem*
  Increasing the RR while keeping the IT the same shortens expiration and may lead to inadequate emptying of lung and intrinsic PEEP.

Ventilator Rate Needed for a Desired $PaCO_2$

Equation 1

$$\text{New rate} = \frac{\text{Rate} \times \text{PaCO}_2}{\text{Desired PaCO}_2} = \frac{(10 \times 50)}{40} = \frac{500}{40} = 12$$

Equation 2

$$\text{New rate} = \frac{(\text{Rate} \times \text{PaCO}_2) \times (V_T - V_D)}{\text{Desired PaCO}_2 \times (\text{New } V_T - \text{New } V_D)}$$

New rate: Ventilator rate needed for a desired $PaCO_2$

Rate: Original rate per minute

$PaCO_2$ = Original arterial carbon dioxide tension in mmHg

Desired $PaCO_2$ = Desired arterial carbon dioxide tension in mmHg

V_T = Original tidal volume

V_D = Original dead space volume

New V_T = Desired tidal volume

New V_D = Desired dead space volume

CHAPTER

22

Ventilator Alarm Settings

Different ventilators will have different alarm systems but the below mentioned are basic alarms which every ventilator should have. Triggering of an alarm is designed to alert the clinician to prevent undesirable technical or patient events.

- Technical events
  Technical events are those events limited to the performance of the ventilator.
- Patient events
  Patient events are those relating to the patient's condition.

ALARM

An absolute value of a parameter on the ventilator beyond which an alert is invoked to warn that the safety limit has been breached. The alarm can be visual, audible or both, depending upon the seriousness of events. Mechanical ventilation must be monitored to avoid harm to critically ill patients.

IMPORTANCE OF VENTILATORY MONITORING

1. To Detect Life Threatening Changes

The primary function of ventilatory monitoring is to detect changes, which could be life threatening for the patient. Acoustic and optical alarms are used to alert ICU staff to ventilatory changes. Once the type of alarm is identified steps can be taken to identify and treat the problem.

- *Sedated or Paralysed Patient*

  The sedated or paralysed patient is extremely vulnerable to failure of the ventilator to deliver the set tidal volume. Alarm on ventilators will warn about any potentially dangerous problems such as disconnection of the patients from the ventilator, apneic episodes and dangerously high airway pressure.

- *Spontaneously Breathing Patient*

  If the patients is breathing spontaneously through the ventilator circuit (CPAP, PSV, ASB) the ventilator can be set to alarm automatically or to provide backup positive pressure breaths if it does not detect spontaneous breath within the specified period.

2. To Recognize Slower Changes to Avoid Critical Situation

A secondary function is to recognize slower changes in respiratory mechanics which give enough time to avoid critical situations.

CLASSIFICATION OF MACHINE MONITORING

1. *Input* power alarms—Logistic alarm
   - Loss of electric power
   - Loss of pneumatic power.
2. *Control* circuit alarms.
3. *Output* alarms
   - Pressure alarms (Ventilation pressure)
     Occlusion alarm—High peak and high mean pressure
     Disconnect alarm—Low peak and low mean pressure
   - Volume alarms—Volume monitoring
     High exhaled tidal volume or high minute volume alarm
     Low exhaled tidal volume—Low minute volume alarm
   - Flow alarm
     Exhaled minute volume
   - Time alarms
     - Respiratory rate
       High frequency or rate alarms—*Tachypnoea monitoring*
       Low frequency or rate alarms—*Apnoea monitoring*
     - Breathing time
       Excessive or inadequate inspiratory time
       Excessive or inadequate expiratory time
4. *Inspiratory* gas alarms
   - Inspired oxygen concentration
   - Inspired gas temperature

5. *Exhaled* gas alarms
    - Exhaled oxygen tension
    - Exhaled end tidal carbon dioxide tension
6. *Machine* malfunctioning—Hard or software

Input Power Alarms—Logistic Alarm

Alarms should be backed up by a battery source to prevent malfunction in the event of electrical failure.

- ***Loss of Electric Power***

  Loss of electrical power or power failure usually results in the ventilator activating a backup alarm that is battery powered. Ventilators typically have alarms that are activated if the electrical power is cut off while the machine is still switched on, for, e.g. if the power cord is accidentally pulled out of the wall socket. Most battery backup alarms are powered by rechargeable nickel cadmium batteries, which are recharged when alternating current (AC) power is available.
    - When AC power is lost, the backup batteries activate audible and visual alarms.
    - If the ventilator is designed to operate on battery power, e.g. transport ventilator there is usually an alarm to warn of a low battery condition.

- ***Loss of Pneumatic Power***

  Ventilators that use pneumatic power have alarms that are activated if either the oxygen or air supply is cut off or reduced below some specified driving pressure. Failure of gas supply either of air or oxygen pneumatic sources will result in a technical event alarm. If either input pressure falls below a specified value from 50 psi, the alarm will result. In some cases the alarm is activated by an electronic pressure switch whereas others are pneumatic reed alarms such as those are incorporated in oxygen blenders.

Control Circuit Alarms

Control circuit alarms alert the clinician for incompatible parameters or failed self diagnostic test.

- Control circuit alarm warns the operator that the set control variable parameters are:
    - Incompatible, e.g. inverse I:E ratio
    - Not within acceptable ranges or specifications

  In the event of an incompatible settings or parameter, the clinician is allowed the opportunity to change the input to one that is compatible.

- Control circuit alarm also warns the clinician that the ventilator has failed some part of a self diagnostic test. This alarm indicates that some aspect of a ventilator self test has failed. In this case, there may be something wrong with the ventilator control circuit itself, for e.g. microprocessor failure, and the ventilator generally responds with a genetic message such as 'ventilator inoperative' and the clinician is alerted by a message display that test failure has occurred.

Output Control Alarm

Output control alarms are those that are activated by an unacceptable state of the ventilator's output. More specifically an output alarm is activated when the value of a control variable (pressure, volume, flow or time) falls outside an expected range.

High and low values may be set for each of these output parameters to alert the clinician of changes in the patient's physiological status.

Pressure Alarms (Ventilation pressure alarm) (Fig. 22.1)

Some ventilators set alarm limits for ventilation pressure automatically and manual adjustment is not required. It is active during mandatory breaths in the ventilatory modes, e.g. IPPV assist, SIMV. Alarm limits for airway pressure are automatically linked to P_{max} and PEEP set values.

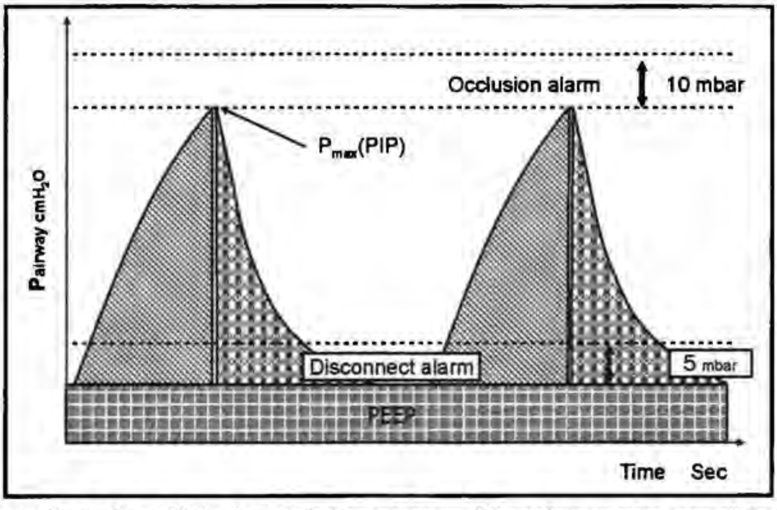


Figure 22.1: Alarm limits for ventilation pressure (For colour version *see* plate 10)

a. *Occlusion Alarm—High Peak Pressure Alarm*

Alarm to be set 10 mbar above P_{max} (PIP) settings

An alarm may be provided to detect failure of the airway pressure to return to the baseline value. A high pressure limit in the breathing circuit serves a dual safety function.

1. It provides an indication of elevated peak airway pressures
2. It also triggers release of excess circuit pressure, thereby protecting the airway from over distension and barotrauma. When excess pressure is reached the ventilator cycles to expiration (pressure control).

Type of ventilatory pressure rise: It can be of the following types:

1. *Intermittent rise:*
   Intermittent rise in peak airway pressure may be due to carinal stimulation, secretions in the tube or high cuff pressure.
2. *Persistent and more gradual rise:*
   Persistent rise in peak pressure indicates a problem with the lung compliance or airway resistance. A more gradual rise can be seen following the deterioration of the patient's pulmonary function (e.g. pulmonary edema), causing lung compliance to decrease progressively, increasing airway resistance.

Reasons of sudden increase in ventilatory pressure: There are various reasons to sudden increase in ventilatory pressure:

1. *Increase in airflow resistance:*
   i *Mechanical factor*
      - Kinking of a breathing circuit/endotracheal tube
      - Ventilator malfunction
      - Cuff herniation
      - Water in circuit
      - Alarm limit set too low

   ii *Patient factor*
      - Main stem bronchial intubation, bronchospasm
      - Tension pneumothorax
      - Decreasing relaxation, coughing
      - Breathing pattern out of a synchronization with ventilator
      - Intraluminal obstruction by secretion - of the tube/tracheostomy.
2. *Decrease in lung or chest wall compliance*
   - Tension pneumothorax
   - Atelectasis
   - ARDS
   - Pneumonia

b. *Disconnect Alarm—Low Peak Pressure*

This alarm is set 5 mbar above end-expiratory settings (PEEP). Low air pressure alarm warns of inadequate pressure in the airline supplying to ventilator and to ensure that a minimum pressure is present in the ventilator circuit during each inspiratory cycle. Low pressure alarm triggering indicates inadequate ventilatory support and not merely system malfunction. This approach will help to minimize the potentially catastrophic airway disasters because an alarm system malfunction should only be considered in the differential diagnosis after immediate life-threatening causes have been thoroughly ruled out by the clinician.

Loss of system pressure

- Power failure
- Source gas failure or disconnection
- Air compressor failure

Loss of circuit pressure

- Loose circuit connection
- Loose humidifier connection
- Circuit disconnection
- Exhalation valve drive line disconnection
- Insufficient ETT cuff volume/cuff deflation

Premature termination of inspiratory phase

- Excessive peak flow
- Insufficient inspiratory time
- Excessive expiratory time
- Inappropriate sensitivity setting (too sensitive)

Inappropriate ventilator settings

- Excessive rate with insufficient peak flow
- Low pressure limit set too high
- Low tidal volume set too high

  If the air source is lost most ventilators will provide 100% oxygen in an attempt to maintain adequate source of gas to the patient.

II. *Volume Alarms—Volume Monitoring*

It includes high/low exhaled tidal volumes for both ventilator supported breaths and spontaneous breaths. Low volumes may result from sedation, disconnection, leaks around the endotracheal tube or apnea and changes in respiratory system mechanics. This alarm may be used in the IPPV, IPPV/assist, SIMV, MMV ventilation modes. It detects and warns when the set tidal volume has not been supplied in following cases:

- When inspiratory flow is too low
- Inspiratory time is short or pressure limit is so low.

a. *Low exhaled tidal volume—Low volume alarm:*

Set volume 100 ml lower than the expired mechanical tidal volume.

Uses

- The volume limit is set to ensure that the patient receives (and exhales) a minimum volume.
- The low expired volume is triggered when the expired volume drops below the preset low volume limit.
- The low volume alarm is usually triggered along with the low inspiratory pressure alarm because loss of airway pressure usually results in loss of volume delivered.

b. *Expiratory minute volume - V_E:*

Expiratory volume measurements provide useful information regarding the patient's ventilatory status (a patient's effective pulmonary ventilation) as well as the functioning of breathing circuit. It is particularly useful to monitor the expiratory minute ventilation when considering adjustments in ventilator for pediatric patients because they may have significant obligatory gas leaks around their uncuffed endotracheal tubes.

High and low values may be set on some ventilators to alert the clinician to changes in the patient's minute volume.

- Upper alarm limit: 20% higher than V_E
- Lower alarm limit: 20 % lower than V_E

Causes high exhaled tidal volume or minute volume alarm

1. Increased respiratory rate
   - Anxiety—Reassure and sedate
   - Pain—Treat pain with analgesic
   - Hypoxia—Evaluate and treat
   - Metabolic acidosis—Correct acidosis
   - Neurogenic hyperventilation
   - Improvement in lung function – Change the settings.
2. Inappropriate or in compatible ventilatory setting
   - Too high tidal volume/respiratory rate
   - Alarm parameters are not set appropriate for prescribed setting V_T/V_E.
3. Ventilator self-cycling

   It may be due to incorrectly set sensitivity—Adjust sensitivity

   Flow trigger—For optimum synchronization of pressure assistance
   - Adult- 3-6 lit
   - Pediatric 1.5-3 lit
   - Neonate 0.75- 1.5 lit

4. Excessive noise (Water from tubing)
   Drainage water from tubing

Time Alarm

High/low ventilator rate alarms alert the clinician to changes in the patient's ventilator rate. The alarm is especially important in the MMV mode and may be used in the BIPAP, SIMV and ASB/Spontaneous.

a. *Rate Alarms*

- *High frequency/or rate alarm—Tachypnoea monitoring:*
  Respiratory rate is monitored to avoid increased dead space ventilation. These alarms indicate hyperventilation or possible machine self triggering. Tachypnoea is a sign of respiratory distress.

  During spontaneous breathing an alarm is given if spontaneus breathing frequency is exceeded for a certain time (alarm time). Frequency is set about 50% above frequency of spontaneous breathing, i.e.10 to 15 breaths per minute over the observed respiratory rate. Set the alarm time for 1 minute.

  A respiratory rate >35/min for a certain period is an indicator of imminent ventilatory insuffiency caused by respiratory muscle fatigue. When the patient consistently sets off the high respiratory alarm, the practitioner may need to increase the level of ventilatory support to ease the patient's work of breathing.

Causes of trigger

- Patient' need to increase ventilation
- Excessive sensitivity
  When this control is set excessively sensitive to the patient's inspiratory effort, minimum inspiratory effort or movement would cause the ventilator to initiate unwanted breaths and increase the total respiratory rate.
- *Low frequency or rate alarms—Apnoea monitoring (Fig. 22.2):*
  Apnoea alarm is high priority alarm. The apnoea alarm is set to ensure that a minimum number of breaths are delivered to the patient. In the spontaneous breathing modes, ASB and CPAP, apnoea monitoring is required. The apnoea alarm should be set with 15-20 second time delay, with less time delay at higher respiratory rate. After apnoea time IPPV breaths are delivered. An alarm is triggered when apnoea occurs usually more than 15 seconds. After further adjustable apnoea time, generally 15 – 60 seconds IPPV breaths are delivered.

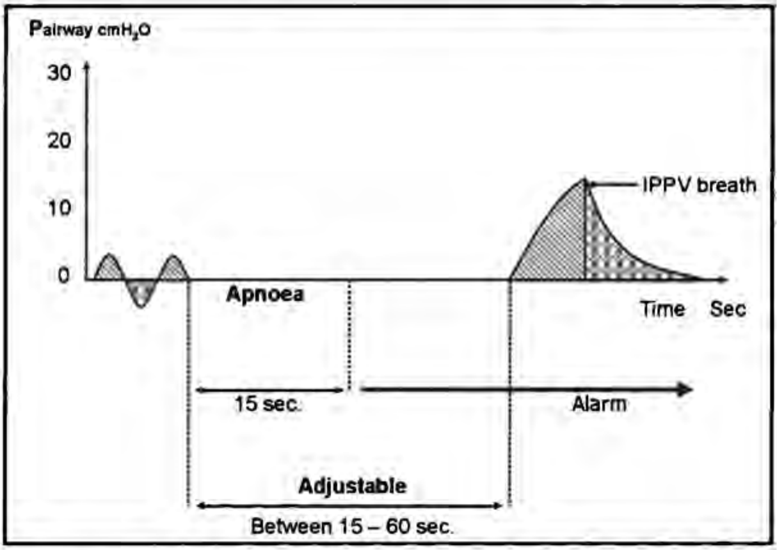


Figure 22.2: Apnoea ventilation alarm (For color version *see* Plate 10)

Period of apnoea:

Adult	20 seconds
Pediatric	15 seconds
Neonate	10 seconds.

Trigger of apnoea:

- Disconnection of the ventilator circuit from the patient's ETT is the most frequent trigger of apnoea alarm, since the ventilator cannot sense any air movement (respiratory effort) from disconnected circuit.
- Respiratory depressants or muscle paralyzing agents
- Conditions of respiratory dysfunction
- Respiratory muscle fatigue

b. *Breathing Time*

Inspiratory and expiratory time alarms may alert the practitioner to circuit obstructions or malfunctions, changes in gas distribution or inappropriate ventilator settings.

- *Inspiratory time*
  1. Excessive inspiratory time
     Inspiratory time that is too long indicates a possible patient circuit obstruction or exhalation manifold malfunction.
  2. Inadequate inspiratory time
     Inspiration time that is too short indicates that adequate tidal volume may not be delivered in pressure controlled mode or that gas distribution in the lungs may not be adequate.

- *Expiratory time*
  1. Excessive expiratory time
     Expiratory time that is too long may indicate apnea.
  2. Inadequate expiratory time
     Expiratory time that is too short may warn of alveolar gas trapping, i.e. expiratory time should be greater than or equal to five time constants of the respiratory system.

Inspired Gas Alarms

Inspired gas alarms alert the clinician to changes in oxygen concentration or gas temperature.

- ***Inspired Oxygen Concentration***

  Some ventilators incorporate an oxygen analyzer to detect changes in FiO_2. High/low alarm alerts the clinician to these changes.

- ***Inspired Gas Temperature***

  Inspired gas temperature may be controlled by a servo-controlled humidifier or monitored by an independent ventilator temperature alarm. High/low temperature alarms can alert the clinician to changes in the inspiratory gas temperature.

Exhaled Gas Alarms

Exhaled oxygen tension or end tidal carbon dioxide tension can be monitored and high/low alarms can be sent to the exhaled gas monitoring system. These monitors can assist the clinician in determining the V_D/V_T gas exchange and determing the respiratory exchange ratio.

- ***Exhaled Oxygen Tension***

  Analysis of end-tidal and mean expired oxygen tension may provide information about gas exchange and could be used along with carbon dioxide data to calculate the respiratory exchange ratio.

- ***Exhaled End Tidal Carbon Dioxide Tension***

  End tidal carbon dioxide monitoring may reflect arterial carbon dioxide tension and thus indicate level of ventilation. Calculation of mean expired carbon dioxide tension along with minute ventilation measurements could provide information about carbon dioxide production and contribute to the calculation of the respiratory exchange ratio and the tidal volume/dead space ratio.

MACHINE MALFUNCTIONING

Hard or Software.

CHAPTER

23

Waveforms of Mechanical Ventilation

Mechanical ventilatory support involves the delivery of flow and pressure to the patient's airway in order to deliver tidal volume. Graphical monitoring of pressure, flow and volume with relation to time helps for optimizing ventilatory support in order to attain goals.

VARIABLES

Pressure, volume and flow are functions of time and are called variables. They are all measured relative to their values at end of expiration.

PARAMETERS

Elastance and resistance are assumed to remain constant and are called parameters.

SCALAR GRAPHICS OR OUTPUT WAVEFORMS OF MECHANICAL VENTILATION

Output waveforms are graphical representations of the control or phase variables in relation to time. Output waveforms are typically presented in the order of pressure, volume and flow. Scalar graphics or waveforms denote changes in variables. These waveforms represent the relationship between the four basic variables of ventilation, i.e. pressure, volume, flow with reference to time. The ventilator determines the shape of the control variables, whereas the other two depend on the patient's compliance and resistance.

How to Read Waveforms

Flow values above the horizontal axis are inspiratory, whereas flow values below the horizontal axis are expiratory. Pressure and volume values rising above the horizontal axis are for inspiration and falling back to the baseline during expiration.

How Graphics Helps

Careful observation and assessment of waveforms during mechanical ventilation can provide useful information for the clinician.

- Waveforms are visual aids which help to identify the changes in lung mechanics and to decide ventilator settings.
- It helps to provide a detailed description of graphic patterns for various modes of ventilation.
- Graphics can be used to understand to optimize mechanical ventilator support.

Types of Waveforms

- Flow time waveform
- Pressure time waveform
- Volume time waveform

PRINCIPLES OF FLOW, PRESSURE AND VOLUME GRAPHICS

Flow Graphic

The flow graphics has two distinctive parts. These are the inspiratory flow graphic and expiratory flow graphic. The inspiratory flow graphic represents the magnitude, duration and flow delivery pattern in the case of a mechanical ventilator breath. For a patient's spontaneous breath it denotes the magnitude, duration and pattern of flow demand.

Inspiratory Flow Graphic (Constant Flow)—Mechanical Breath (Fig. 23.1)

1. *Exhalation valve closes and inspiration begins:*
   It represents the initiation of flow from the ventilator. This can be a time trigger or a patient trigger (flow or pressure).
2. *Peak inspiratory flow rate:*
   This represents the highest inspiratory flow.
3. *Exhalation valve opens and beginning of expiration:*
   This represents the end of inspiration and cessation of flow delivery. The cycling to expiration can be due to volume or time cycle.

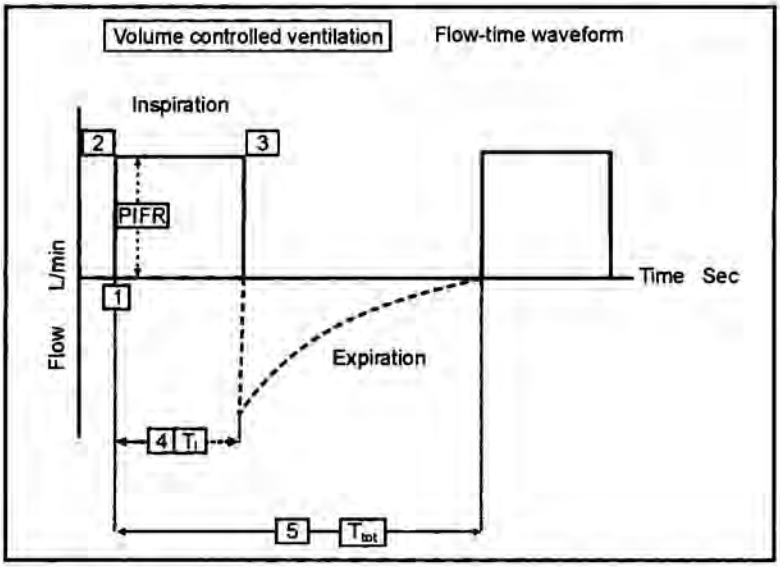


Figure 23.1: Inspiratory flow graphic (constant flow) – Mechanical breath

4. *Inspiratory time* T_i:

   Inspiratory time represents the duration of inspiratory flow. Inspiratory time is determined by preset volume, peak flow and flow pattern. Under some circumstances, inspiratory time is more than duration of flow, when an inspiratory pause is used.

5. *Total breathing time or total cycle time (TCT) or* T_{tot}:

   Total cycle time is determined by preset ventilator rate.

$$T_{tot} = \frac{60\ \text{sec./min.}}{\text{Rate (breath/min.)}}$$

Expiratory Flow Graphics—Mechanical Breath (Fig. 23.2)

Expiration is generally a passive manoeuver, whether from a mechanical breath or spontaneous breath. The magnitude, duration and pattern of expiratory flow graphics are determined by the compliance and resistance of airways and patient circuit (size and length of endotracheal tube, resistance of exhalation valve). Neurological disorders may result in active use of the respiratory muscles during the expiratory phase.

6. *Exhalation valve opens and expiration begins*
7. *Peak expiratory flow rate:*

   This represents the peak expiratory flow. Expiratory flow graphics is same for mechanical and spontaneous breath. However, mechanical delivered tidal volume is generally larger than spontaneous tidal volume, peak expiratory flow of mechanical breath will be higher than for spontaneous breath.

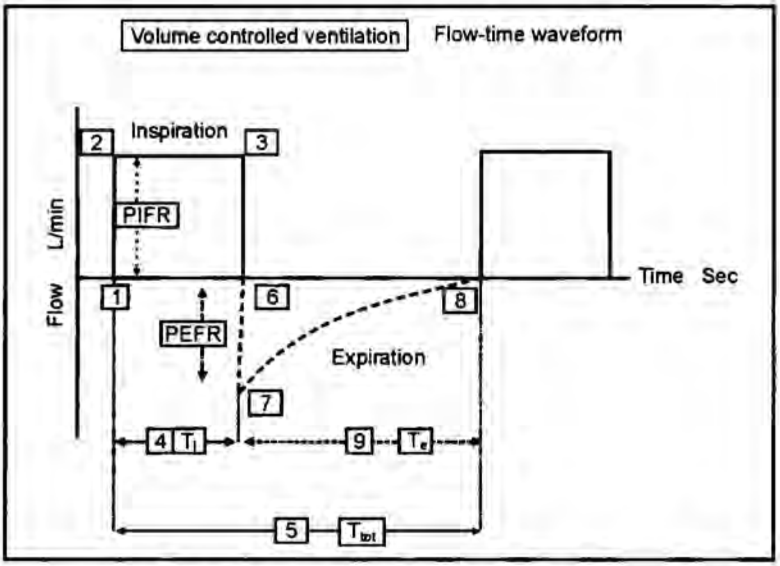


Figure 23.2: Expiratory flow graphics—Mechanical breath

8. *Exhalation valve closes and inspiration begins:*
   This represents end of expiratory flow delivery. The end of the expiratory flow and the start of the next mechanical breath, this point helps to asses the inspiratory to expiratory ration (I:E ratio) and potential for air trapping.
9. *Expiratory time - T_i :*
   Expiratory time represents duration of expiratory flow. Increased resistance due to bronchospasm or accumulation of secretions in the airway (obstructed airway) may result in decreased peak expiratory flow and longer duration of expiratory flow.

Inspiratory Flow Graphics – Spontaneous Breath (Fig. 23.3)

1. Represents the start of inspiration.
2. Represents the magnitude of inspiratory flow
3. Represents the end of inspiration
4. Inspiratory time—Represents the duration of inspiratory flow.

Pressure Graphics

Pressure graphics for mechanical breath and spontaneous breath are distinctly different, and has several components.

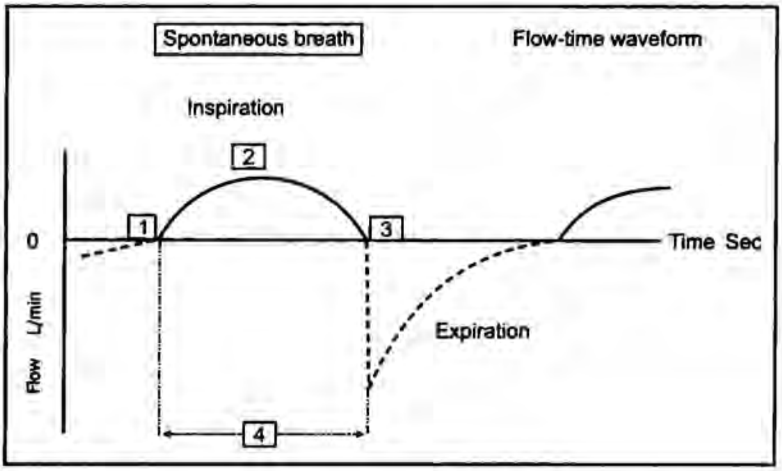


Figure 23.3: Inspiratory flow graphics – Spontaneous breath

Pressure Time Waveform—Mechanical Breath (Fig. 23.4)

1. Represents the peak inflation pressure or peak inspiratory pressure, and is determined by the patient's compliance and resistance as well as the delivered tidal volume and flow rate.
2. Represents the inspiratory time – T_i
3. Represents the duration of positive pressure.

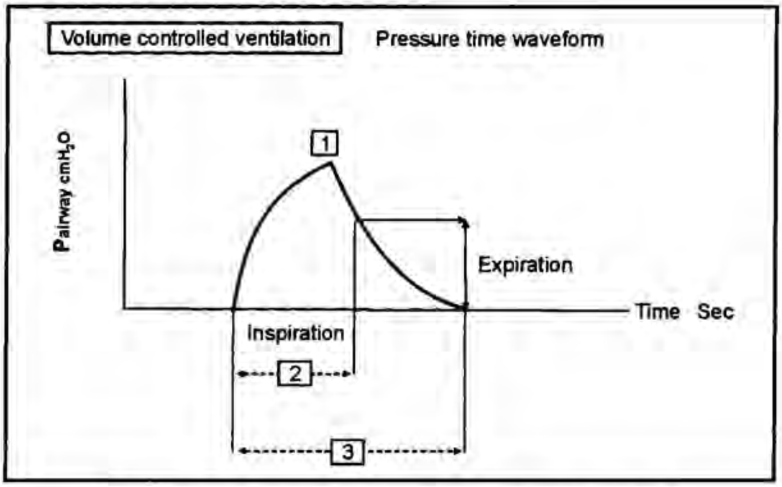


Figure 23.4: Pressure time waveform – Mechanical breath

Pressure Time Waveform – Spontaneous Breath (Fig. 23.5)

1. Represents the pressure drop during inspiration. The magnitude of pressure drop depends on the patient's peak inspiratory flow rate and trigger sensitivity.

Represents the minimal pressure rise during the expiratory phase. The increase in pressure is due to flow resistance in the expiratory limb. The magnitude of the pressure increase will vary with the peak expiratory flow rate. Because peak expiratory flow rate increases when the respiratory muscles are active during the expiratory phase, the pressure may rise more dramatically if the patient is actively or forcibly exhaling. Additionally, high levels of continuous flow running through the ventilator circuitry may also increase the pressure rise during expiration.

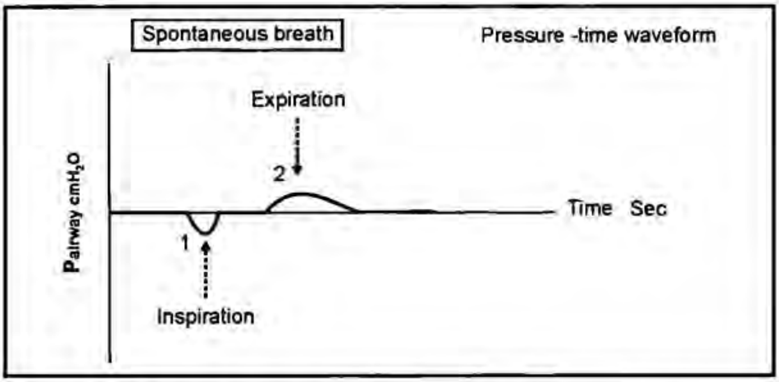


Figure 23.5: Pressure timewave form – Spontaneous breath

Components of Inflation Pressure (Fig. 23.6)

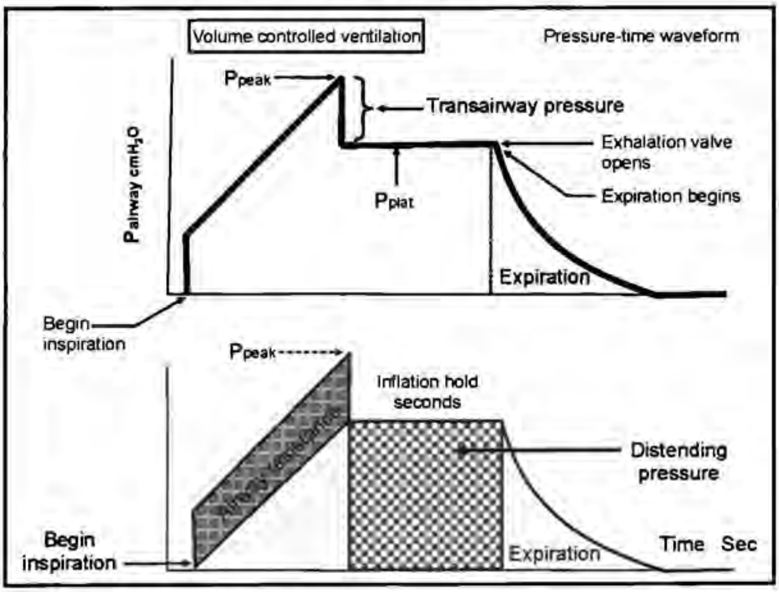


Figure 23.6: Components of inflation pressure

The term inflation pressure refers to the pressure required to a deliver tidal volume. Inflation pressure has two components, flow resistive pressure and lung distending pressure.

Peak inspiratory or peak airway pressure – PIP / PAP / P_{peak}

Peak airway pressure represents the maximum pressure applied to the proximal airway which will affect flow and volume changes. P_{peak} pressure is used to deliver the tidal volume by overcoming non-elastic (airways) and elastic (lung parenchyma and chest wall) resistance. PIP represents the peak dynamic pressure. Dynamic characteristics thus reflect the resistive and elastic properties of the respiratory system.

Plateau/Alveolar pressure – $P_{Plateau}$ /$P_{alveolar}$ pressure

$P_{Plateau}$ /$P_{alveolar}$: pressure is the pressure needed to maintain lung inflation [Elastic (lung and chest wall parenchyma) resistance] in the absence of airflow. Plateau pressure represents total static compliance. Static compliance reflects the elastic properties of the respiratory system. It is often referred to as a close estimate of the alveolar pressure which is the pressure during the pause time.

Pressure Time Waveform in Volume Controlled Ventilation (Fig. 23.7)

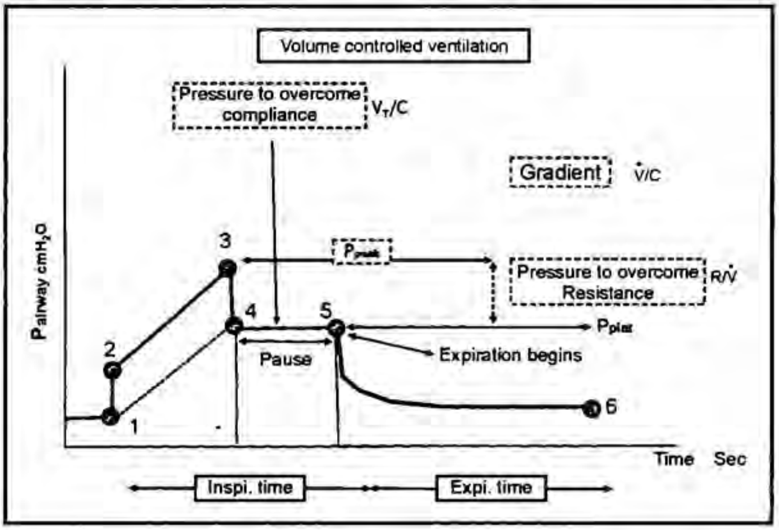


Figure 23.7: Pressure time waveform

At the beginning of inspiration the pressure between point 1 and 2 increases on account of the resistance in the system. The level of the pressure at point 2 is equivalent to the product of resistance and flow.

$$\Delta P = R \times \dot{V}$$

After point 2, the pressure increases in a straight line till point 3 – which is peak pressure. The gradient of this curve is dependent on the inspiratory flow and the overall compliance.

Inspiratory flow $\dot{V}$/Compliance C

At point 3 the ventilator delivers the set tidal volume and no further flow is delivered. Because of no flow at this point 3 the pressure falls to plateau pressure. This drop in pressure is equivalent to the rise in pressure caused by the resistance of the system at the beginning of inspiration called transairway pressure, i.e 1and 2 = 3 and 4.

There may be a slight decrease in pressure between points 4 and 5. Lung recruitment and leaks in the system are the possible reason for this. The plateau pressure is dependent on the compliance and tidal volume. During the plateau time no volume is supplied to the lung and the inspiratory flow is zero. At point 5 expiration begins because of the elastic recoil forces of the chest and is a passive process.

CLINICAL SIGNIFICANCE OF VARIOUS WAVEFORMS IN VOLUME CONTROLLED VENTILATION

Pressure Time Waveform

Ventilator settings such as flow and inspiratory time, regulate how steep the waveform rises toward peak inspiratory pressure. In the pressure-time waveform the airway pressure is plotted against time. The pressure is expressed in cm H_2O and the time in seconds. The curve shows the gradual changes in airway pressure with time.

1. To diagnose progress or worsening lung mechanics with the help of components of inflation pressure:

   The various components of the pressure versus time curve if studied carefully helps to diagnose progress or worsening lung mechanics

   - *Change in airway resistance (Fig. 23.8)*
     PIP during mechanical ventilation depends on resistance, compliance, inspiratory flow, tidal volume. When plateau pressure doesn't change but peak pressure increases it denotes an increase in airway resistance. A large pressure increase from either large inspiratory flow or high resistance

   - *Change in compliance (Fig. 23.9)*
     The trans-airway pressure and the length of the plateau (inflation hold) will differentiate between increase in R_{airway} (airway resistance) and decrease in compliance, i.e. change in P_{alv} (alveolar pressure). The contribution of resistance and elastance to the peak inspiratory pressure can be distinguished by occluding the expiratory tubing at the end of inspiration.

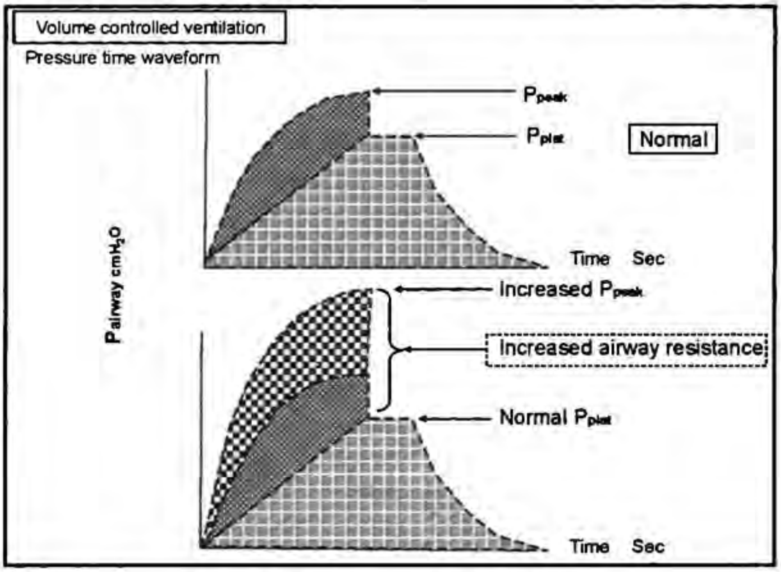


Figure 23.8: Increased peak pressure

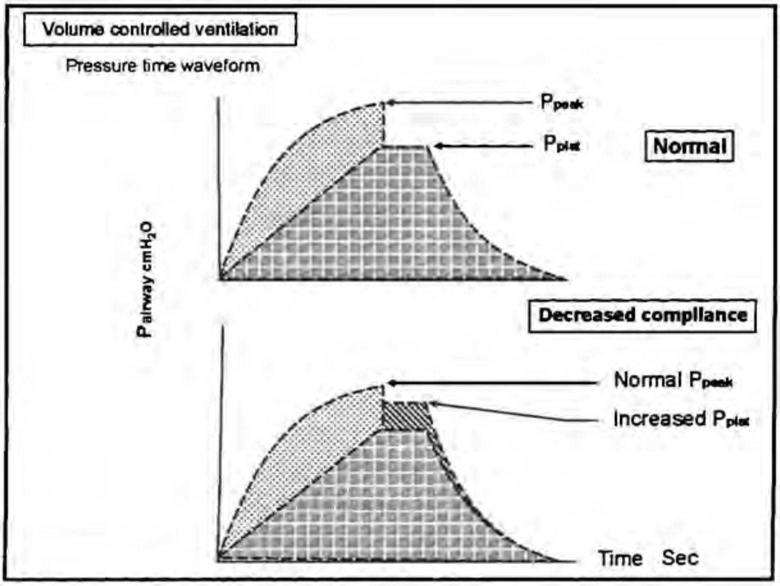


Figure 23.9: Increased plateau pressure

When the inflation volume is held in the lungs occluding the tracheal tube or ventilator tubing to prevent lung deflation, the proximal airway pressure decreases initially and reaches a steady level, which is called the end-inspiratory plateau pressure. Because no airflow is present when the plateau pressure is created, the pressure is not a function of flow resistance in the airways. Instead, the plateau pressure ($P_{plateau}$) is directly proportional to the elastance of the lungs and chest walls.

2. High inspiratory flow helps to prolong expiratory time: (Fig. 23.10)
   High inspiratory flow decrease inspiratory time and prolongs expiratory time provided there is no change in the respiratory rate.

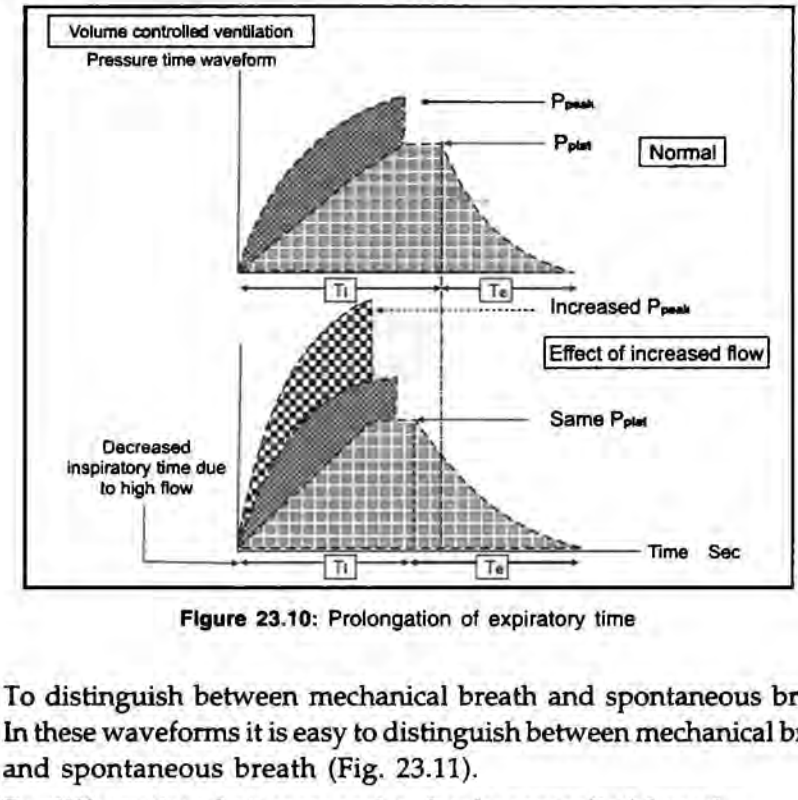


Figure 23.10: Prolongation of expiratory time

3. To distinguish between mechanical breath and spontaneous breath:
   In these waveforms it is easy to distinguish between mechanical breath and spontaneous breath (Fig. 23.11).
4. To differentiate between assisted v/s controlled breath:
   In the pressure versus time waveform the triggering mechanism can be easily seen, which helps to differentiate between assisted v/s controlled (Fig. 23.12).
5. To differentiate between adequacy or inadequacy of inspiratory flow (Fig. 23.13):
   The shape of pressure time waveform can help to differentiate between adequacy or inadequacy of inspiratory flow.

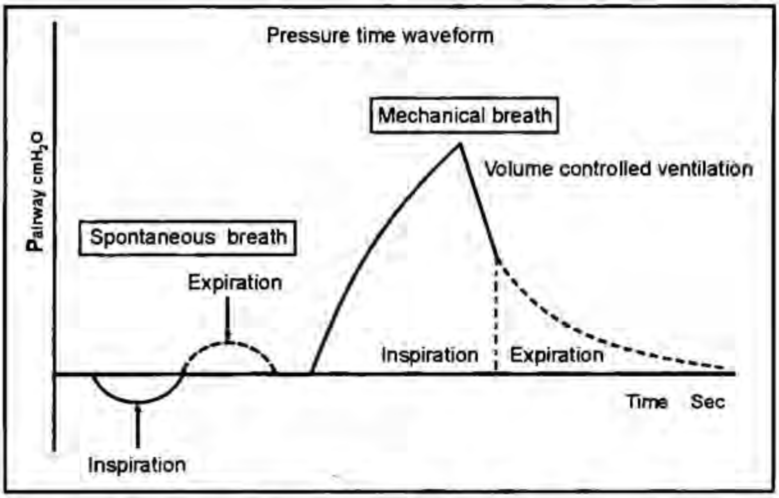


Figure 23.11:To distinguish between spontaneas breath and mechanical breath

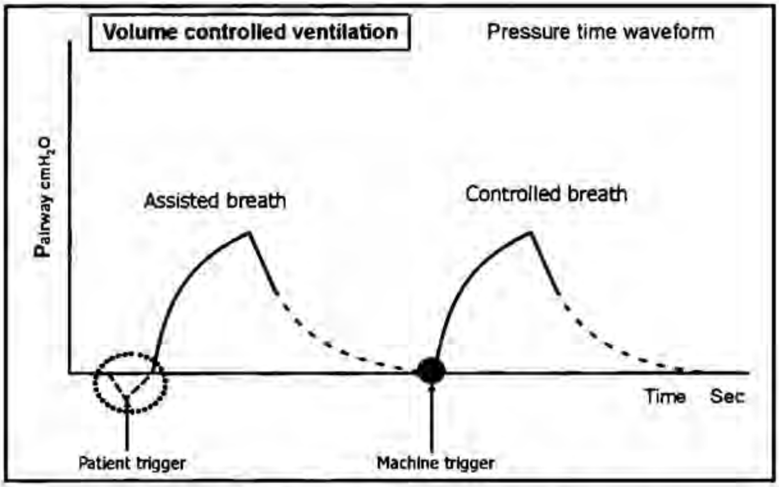


Figure 23.12: Triggering mechanism can be seen

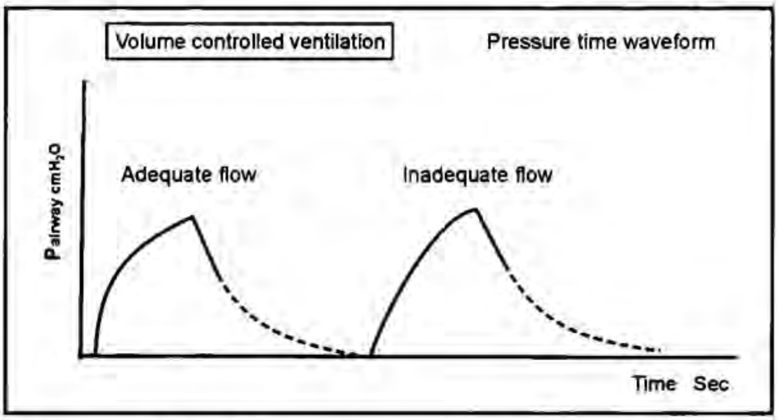


Figure 23.13: Adequacy or inadequacy of inspiratory flow

Increased Usefulness in Comparing Three Graphics

Flow, volume and pressure waveforms are individually useful; however their usefulness increases if all three waveforms are used in conjunction with each other.

Relative Orientation of Graphics Helps to Differentiate Mode of Ventilation

The pattern of flow, pressure, and volume delivery to a patient is termed the mode of ventilatory support. Each mode has its own characteristic flow, pressure, volume waveforms.

Volume-controlled Ventilation with Constant Flow (Figs 23.14 and 23.15)

In volume-controlled ventilation, constant flow and tidal volume are preset and the height and course of the airway pressure (variable pressure)in the inspiratory phase depends on the airway resistance and compliance properties of the respiratory system.

Pressure-controlled Ventilation (Figs 23.16 and 23.17)

Pressure increases rapidly from lower pressure until it reaches the pre set pressure and then remains constant for the set inspiration time. The drop in pressure during the expiratory phase follows the same curve as in volume-controlled ventilation.

As the pressure is preset, pressure time waveform shows either no change or very minute change, as a consequence of changes in resistance and compliance.

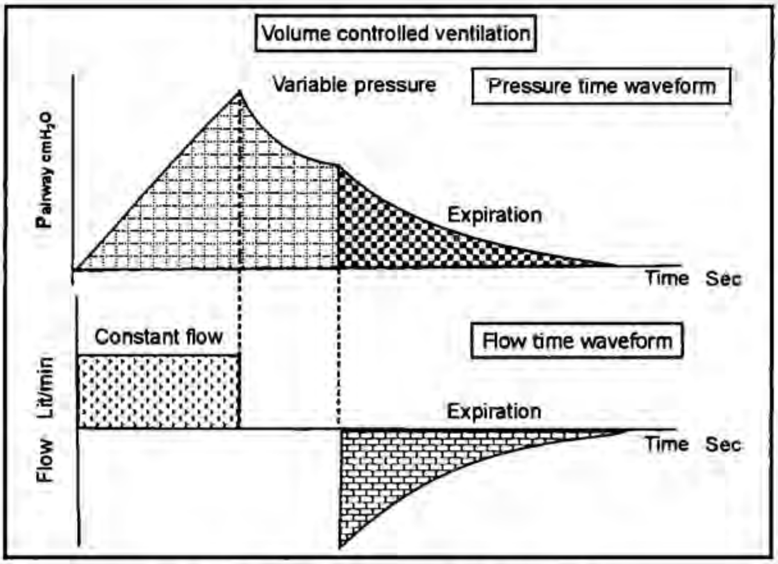


Figure 23.14: Variable pressure and constant flow

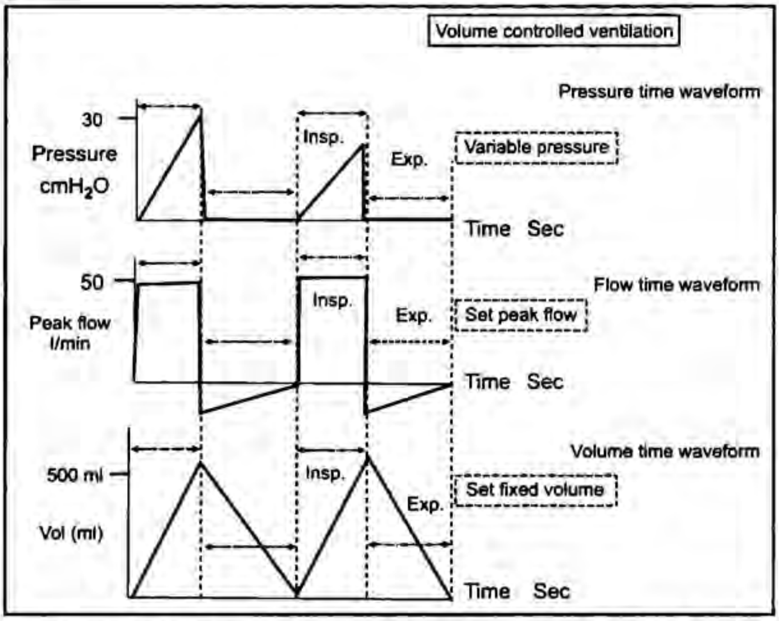


Figure 23.15: Variable pressure, constant flow and constant volume

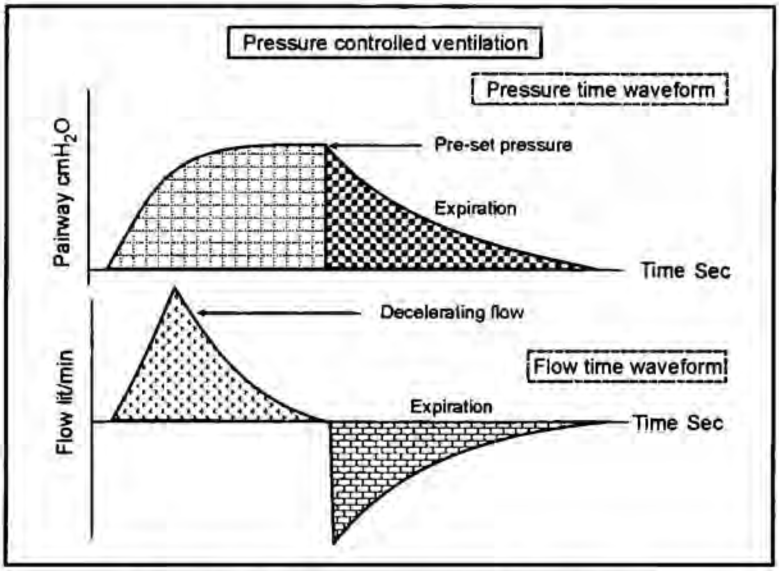


Figure 23.16: Constant pressure and variable flow

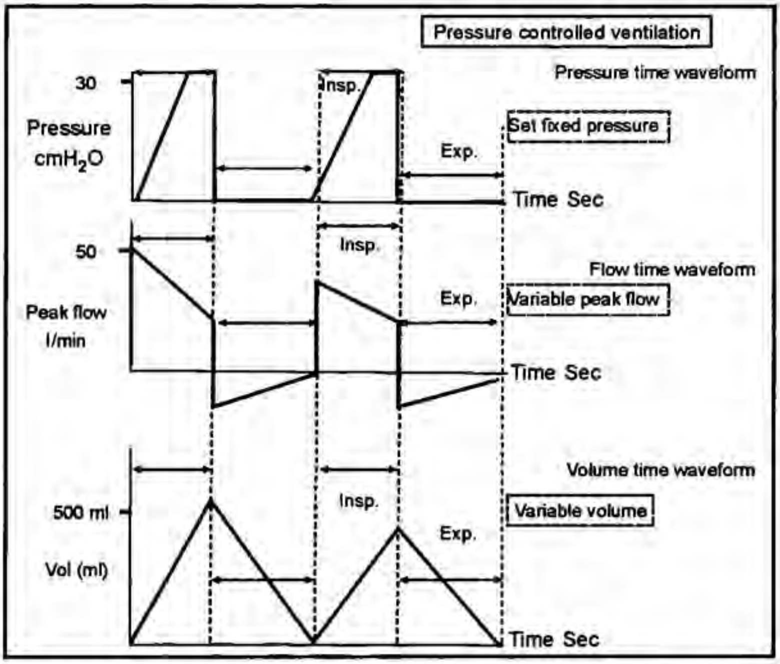


Figure 23.17: Constant pressure, variable flow and variable volume

Flow Time Waveform

Flow time waveform shows the gradual changes in the inspiratory and expiratory flows with time. During inspiration the course of the flow curve is dependent on, or at least strongly influenced by, the mode of ventilation set on the ventilator. The most common types of flow are constant flow and decelerating flow.

1. To differentiate mode of ventilation depending on flow pattern (Fig. 23.18).

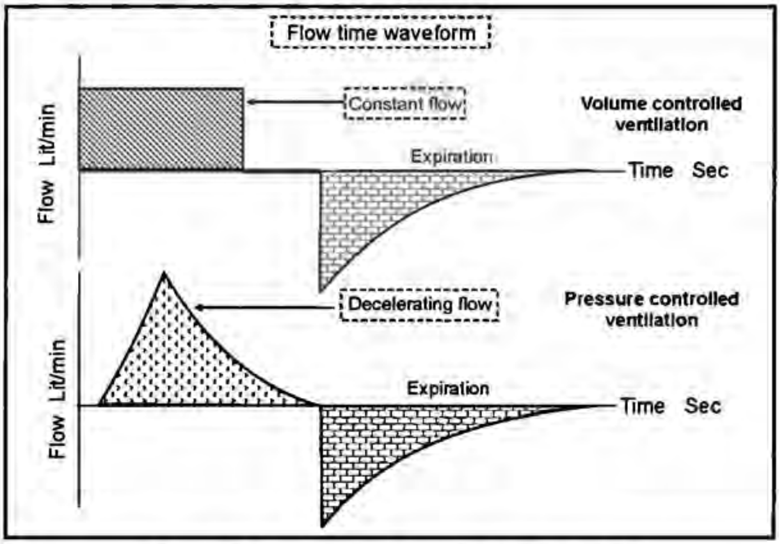


Figure 23.18: Flow time waveform in VCV/PCV

   - The constant flow pattern is a typical feature of volume controlled ventilation.
   - Decelerating flow is a typical feature of pressure controlled ventilation.

2. It is easy to distinguish between spontaneous breath and mechanical breath in flow time waveform (Fig. 23.19)
3. Volume controlled ventilation showing high flow (Fig. 23.20)
   The inspiratory flow is a measure for the velocity with which the breathing gas is delivered.

   If ventilation occurs with a high inspiratory flow, then preselected tidal volume is delivered before the inspiratory time is completed and then inspiratory time can be divided into a flow and no-flow phase. In the pressure-time waveform of volume controlled ventilation with high inspiratory flow, an inspiratory pressure plateau (no flow phase) will be seen.

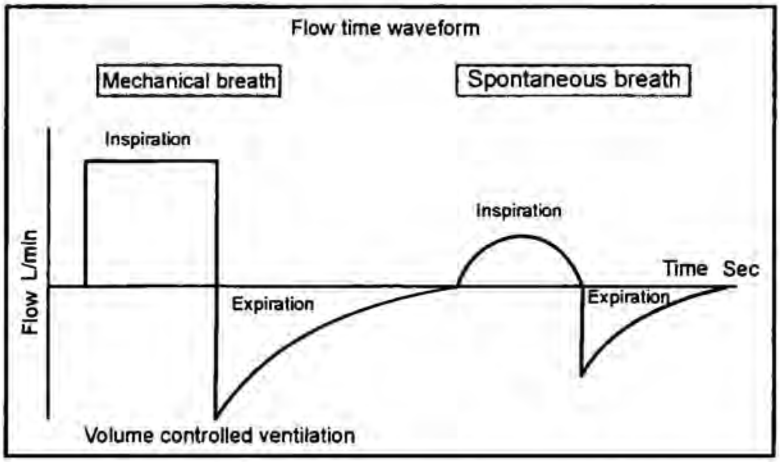


Figure 23.19: Mechanical breath and spontaneous breath

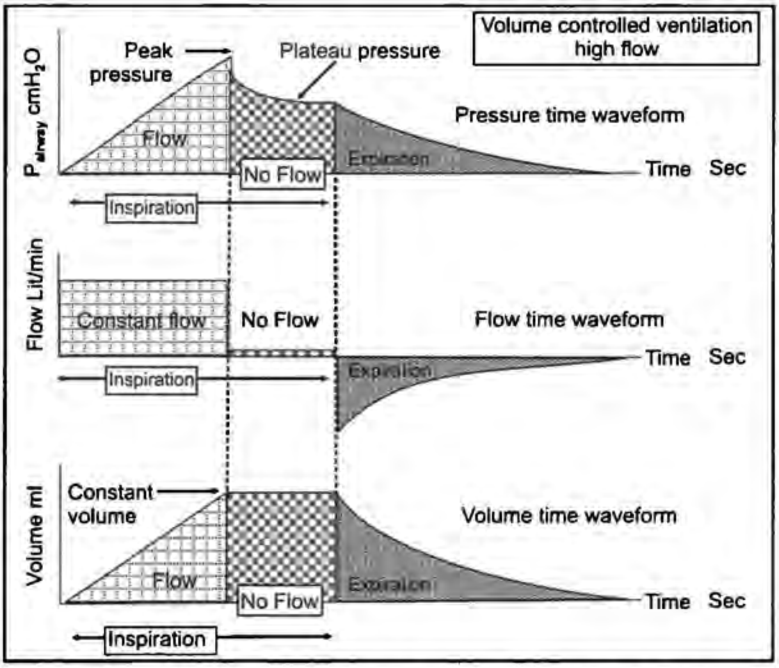


Figure 23.20: Pressure/flow time waveform showing high flow

4. Flow time waveform helps to diagnose increased expiratory resistance and to evaluate response to bronchodilator (Fig. 23.21)

A more gentle expiratory flow curve indicates increased expiratory resistance. It also aid in the assessment of bronchodilator therapy during mechanical ventilation

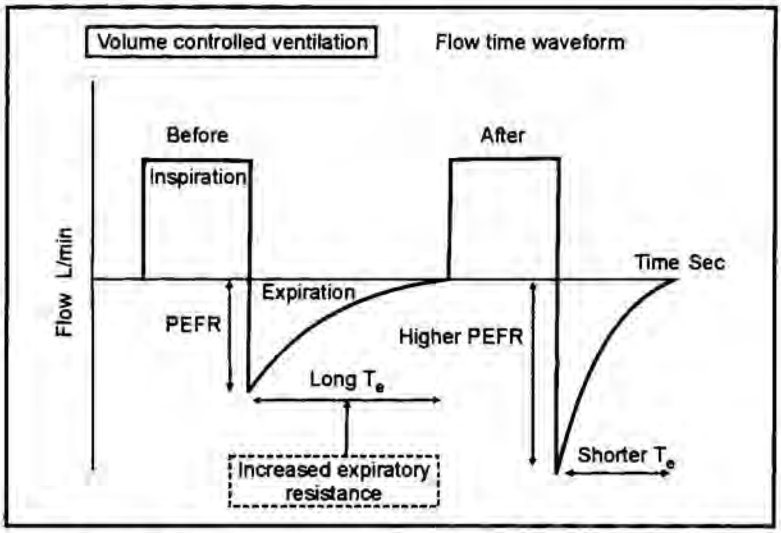


Figure 23.21: Waveforms showing response to bronchodilators

5. Flow time waveform in volume controlled, it is easy to diagnose air leaks (Fig. 23.22)

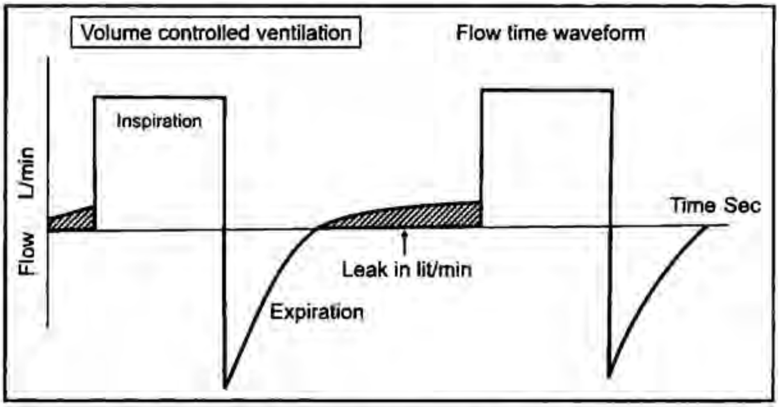


Figure 23.22: Wareforms showing air leaks

6. Flow time waveform is very useful in diagnosing auto-PEEP
   - If the flow does not return to zero during expiration (insufficient time for expiration), it indicates the presence of air trapping giving rise to auto-PEEP (Fig. 23.23)
   - Flow does not return to baseline during a passive expiration since expiratory time constant is shorter than required time constant. The required time constant is three time constant (Fig. 23.24)

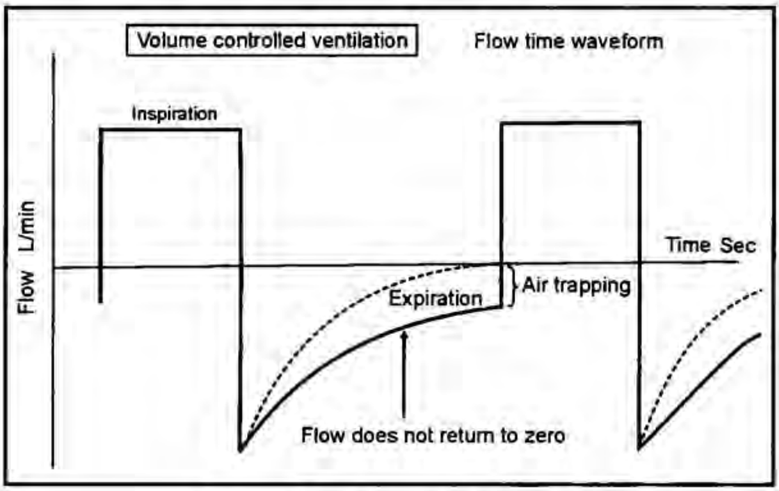


Figure 23.23: Presenic of auto-PEEP

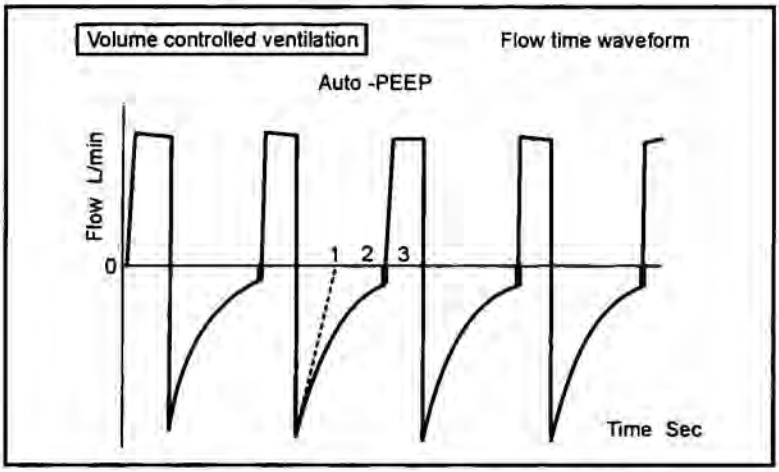


Figure 23.24: Expiratory time constant shorter than 3τ

Volume Time Waveform

Volume time waveform is the gradual changes in the volume in the alveoli during the inspiratory and expiratory time.

1. Volume changes in volume time waveform
   During the inspiratory flow phase, the volume increases continuously, during the pause it remains constant and during expiration the volume decreases as a result of passive expiration (Fig. 23.25).
2. Detection of air leaks (Fig. 23.26)
   Air leaks can be detected when exhalation does not return to zero, i.e. volume exhaled is less than the volume delivered or inhaled.

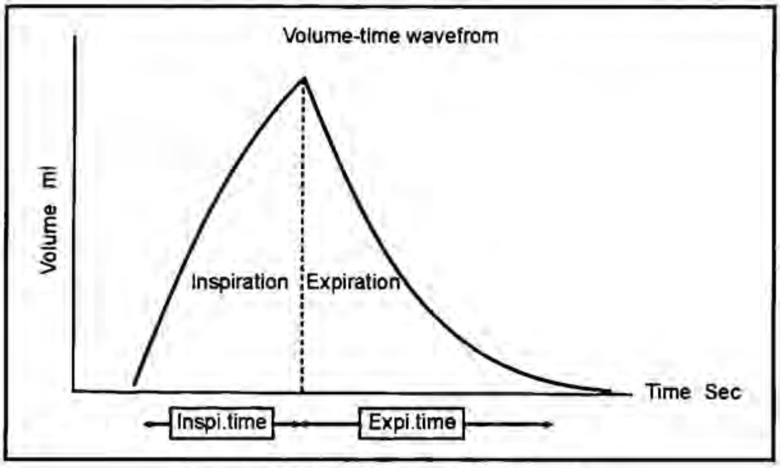


Figure 23.25: Volume changes are seen

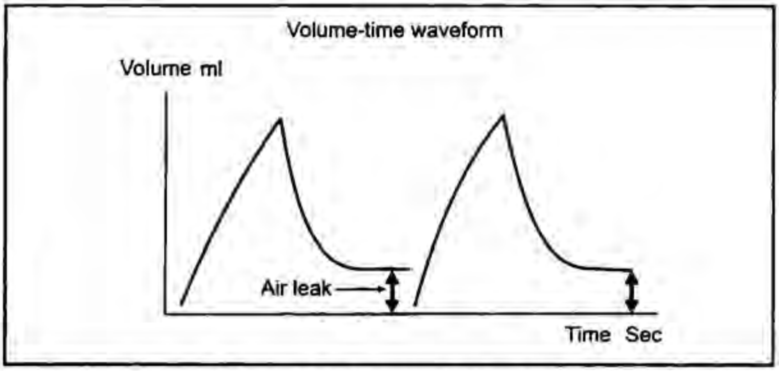


Figure 23.26: Presenic of air leak

3. Detection of active exhalation (Fig. 23.27)
   Active exhalation by the patient can be detected when the patient is breathing while on ventilator.

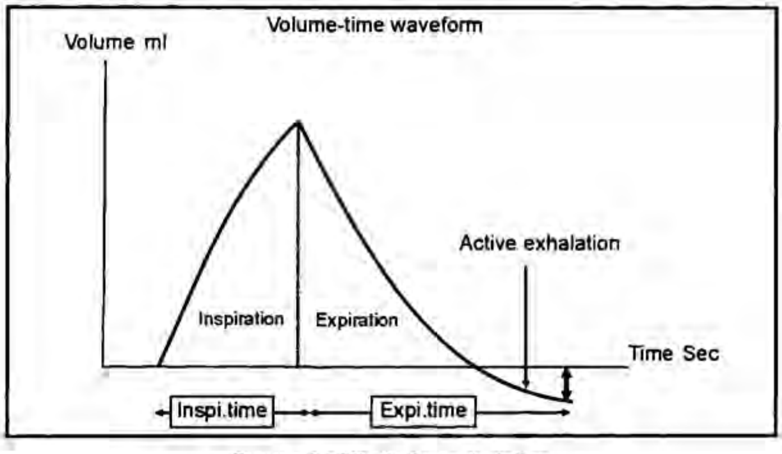


Figure 23.27: Active exhalation

The Change from (VCV) to PCV Resulted in More Efficient Elimination of CO_2 per Breath (Fig. 23.28)

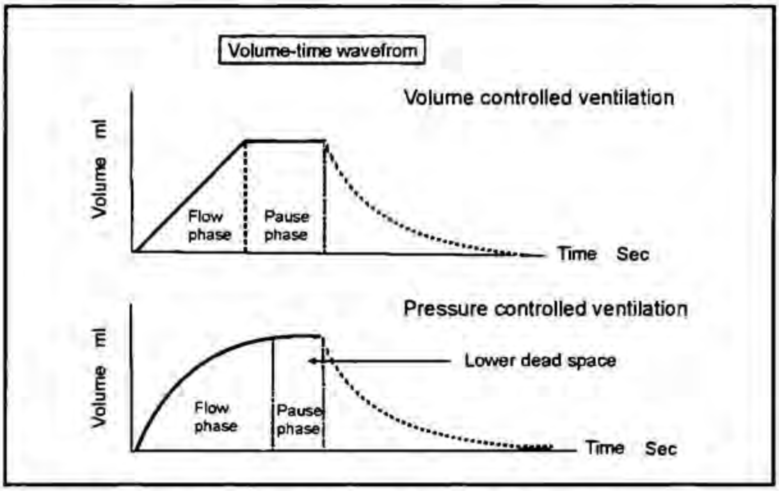


Figure 23.28: The change from VCV to PCV resulted in reduction in pause time

The change from flow controlled, volume cycled ventilation (VCV) to PCV while keeping the same inspiratory time, resulted in more efficient elimination of CO_2 per breath, reflecting a lower dead space.

Leak Compensation with Change in Flow (Fig. 23.29)

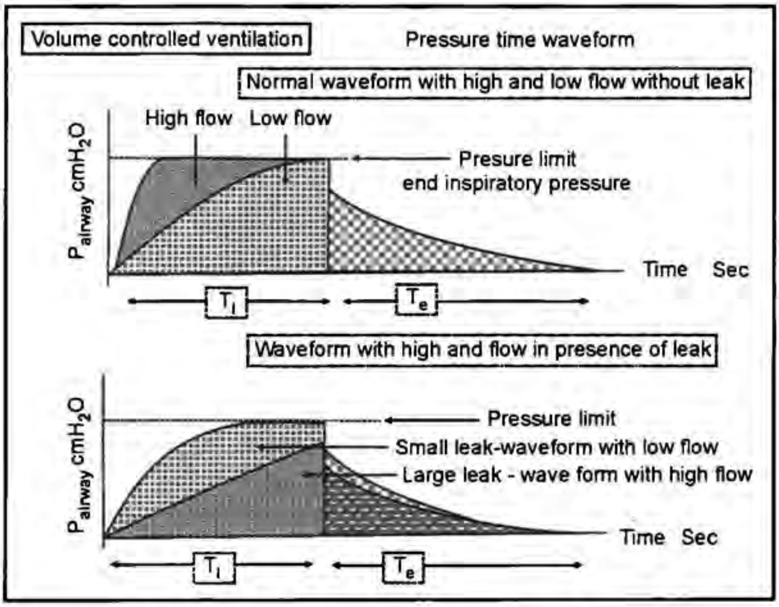


Figure 23.29: Leak compensation for small leaks

Leaks in the breathing system or around the endotracheal tube leads to a steep decrease in tidal volume, particularly in volume constant ventilation. By increasing the inspiratory flow, constant flow respirators can compensate for small to medium leaks.

With larger leaks, the set inspiratory pressure is not reached, despite sufficiently high flow, and a reduced tidal volume is the result. Ventilation is sufficient as long as an inspiratory plateau remains.

CHAPTER

24

Time Constants in Mechanical Ventilation

WASH-IN CURVE

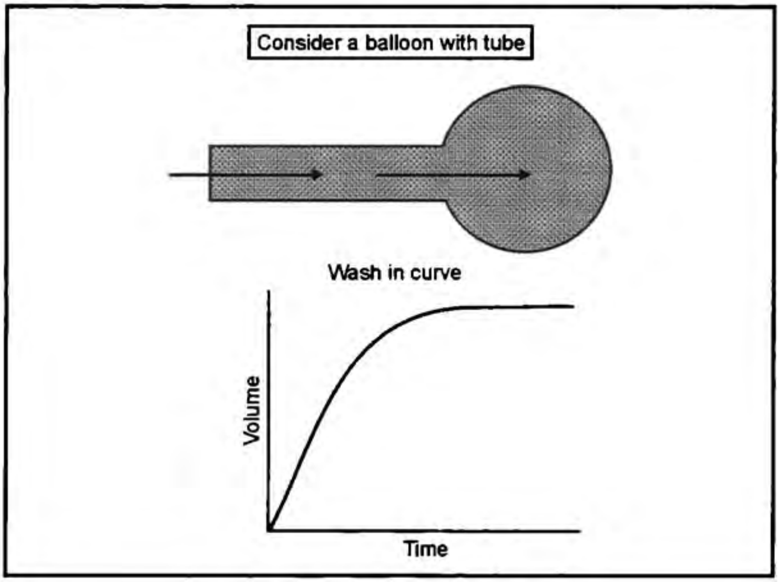


Figure 24.1: Wash in curve

Consider a balloon with a tube leading to it. The inflation of the balloon in response to a given inflationary pressure is described by the exponential 'wash-in curve' (Fig. 24.1).

FAST AND SLOW ALVEOLI (FIG. 24.2)

A short inspiratory time causes poor distribution of the inspired gas due to the characteristics of 'fast' and 'slow' alveoli.

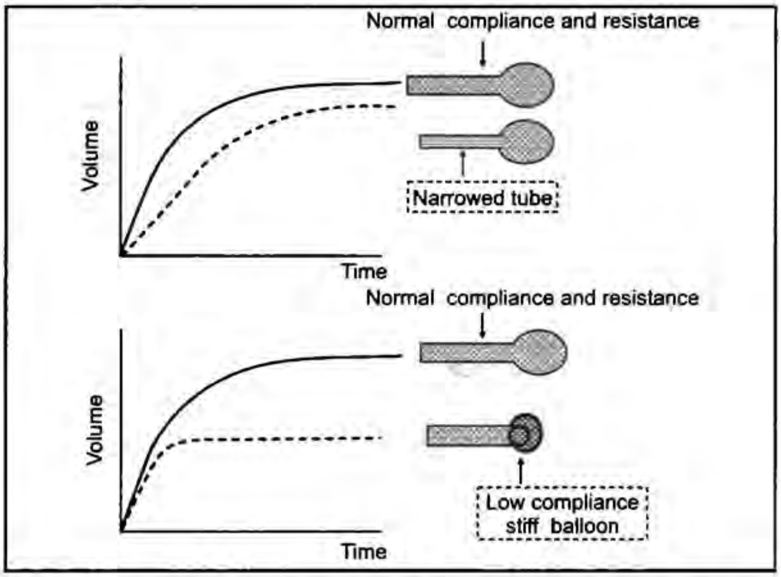


Figure 24.2: Narrowed tube and Low compliance (stiff) of the balloon

Narrowed Tube

If the tube leading to the balloon is narrowed the balloon will still reach the same final volume when inflated at the same pressure but it will take longer to do so.

Low Compliance (stiff) of the Balloon

The compliance of the balloon is also of importance. If the wall has a low compliance (stiff) the balloon will reach a smaller final volume when subjected to the same inflation pressure, and this volume will be reached after a shorter time

EXPONENTIAL CURVE

The exponential curve can be described in terms of its time constant (y) (Fig. 24.3).

Time constant (time interval) is the time it would take for the balloon to reach its final volume if the initial gas flow is maintained through inflation. What has been said about the balloon can be applied to the lungs.

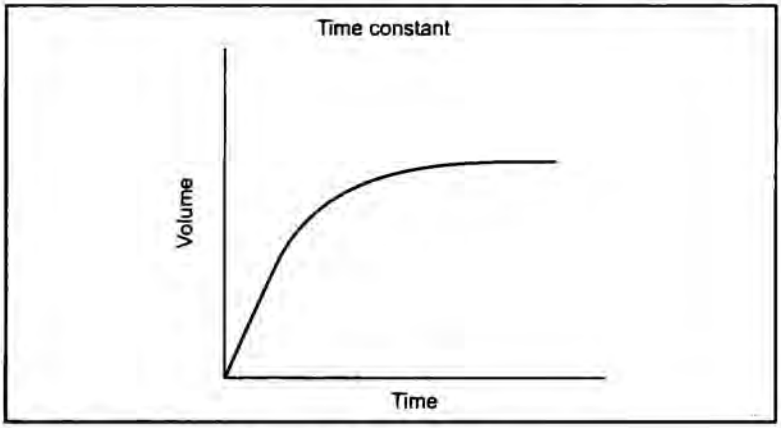


Figure 24.3: Exponential curve

COMPARTMENT

A group of identical breathing mechanical subunits of the lung is referred to as a 'Compartment'

Compartment Model of the Lung

The simplified model of lung mechanics consists of two separate components.

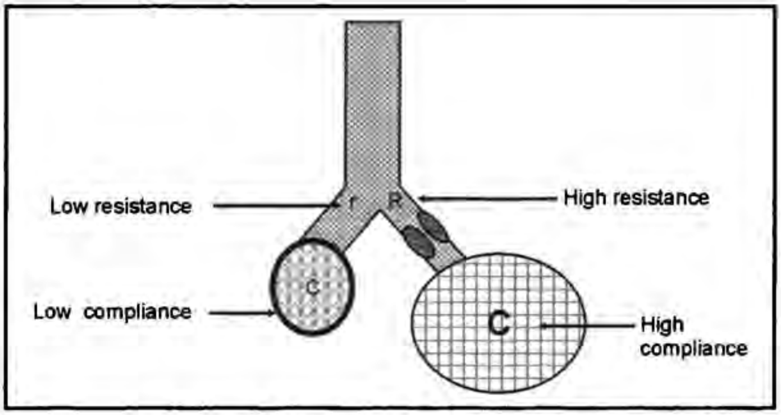


Figure 24.4: Compartment model of the lung

1. *Resistance:* The resistance represents the flow resistance of the relevant lung compartment.
2. *Compliance:* Compliance describes regional elasticity.

In order to achieve gas flow into the lungs, resistive and compliant forces in the lung must be overcome. It is important to understand the concept of a time constant.

Inspiratory and expiratory time constants are very close in normal value in a normal lung but their differences may be accentuated in various disease states. In addition, different segments of the lung may have different time constants. A lung compartment consists of a combination of resistance and compliance and has a time constant, which arises from the product of two dimensions.

$$\tau = R \times C$$

- The time constant Greek letter τ (tau) expresses how quickly a compartment can react upon an alteration of pressure.
- It is a measure of how quickly an alveolar unit reaches equilibrium (fills or empties).
- It is therefore a measure of the filling or emptying velocity.

Clinical Applications of Time Constants

- Time constant is a measure of how quickly pressures in the proximal airways (or ventilator tubing) can be delivered or equilibrated in the alveoli.
- It also reflects how quickly a lung can exhale after having been inflated.

EXPONENTIAL FUNCTIONS

The respiratory system, like most biological systems, is closely associated with exponential functions. An exponential function is a mathematical expression that describes an event where the rate of change of one variable is proportional to its magnitude.

Expiratory Flow in a Passive Breath

In a passive breath, expiratory flow will be higher at the beginning of expiration than at the end, as the lung volume decreases towards the functional residual capacity (FRC).

There are various forms of exponential functions but the most important ones or the clinician in mechanical ventilation are:

1. Rising exponential functions
2. Decaying exponential functions

Rising Exponential Function

A rising exponential function expresses as an increase of one variable as a function of time.

- Volume vs. time
- Flow vs. time
- Pressure vs. time

In mechanical ventilation with a constant pressure mode, the inspiratory volume-time waveform is an example of a rising exponential function.

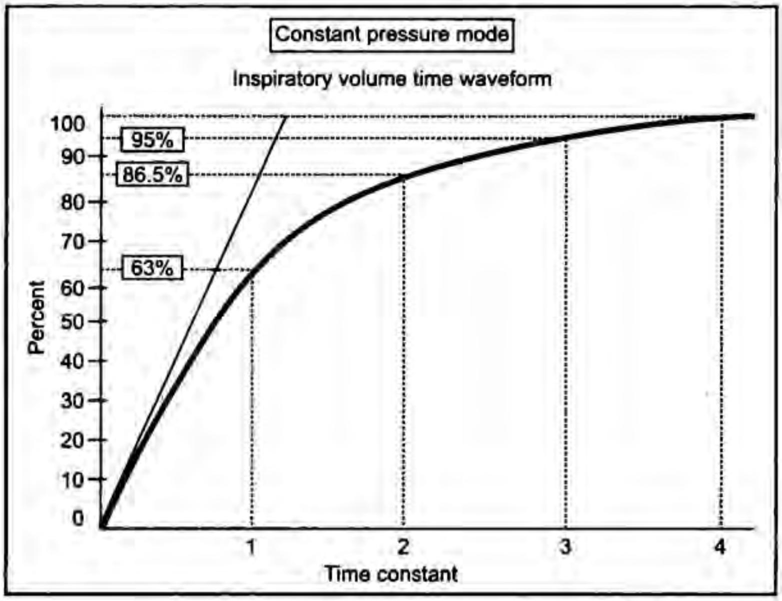


Figure 24.5: Rising exponential function

Figure 24.5—A rising exponential function expresses the behavior of a physical system where the rate of change of one variable is proportional to its magnitude and a constant

In a physical system, the constant is usually the final value of the variable studied. In this expression, as time progresses and the function y approaches its final value (y final) the rate of change decreases towards zero. The difference between the initial value of y and its final value is always largest at the beginning of the event. Therefore the largest rate is always observed at the beginning of the event and the smallest rate of change at the end of the event. As y approaches its final value (y final) – the difference between y final – y will approach zero and the rate of variation of the variable y will be very small.

t – is the period of the time after the onset of the event

y – is the value of the variable at time t

y final – is the final value of y

Decaying Exponential Function

A decaying exponential function of a decrease of one variable as function of time.

- Volume vs. time
- *Flow vs. time (Fig. 24.6):*
  Flow return to baseline during a passive expiration is an example of a negative decaying exponential function.

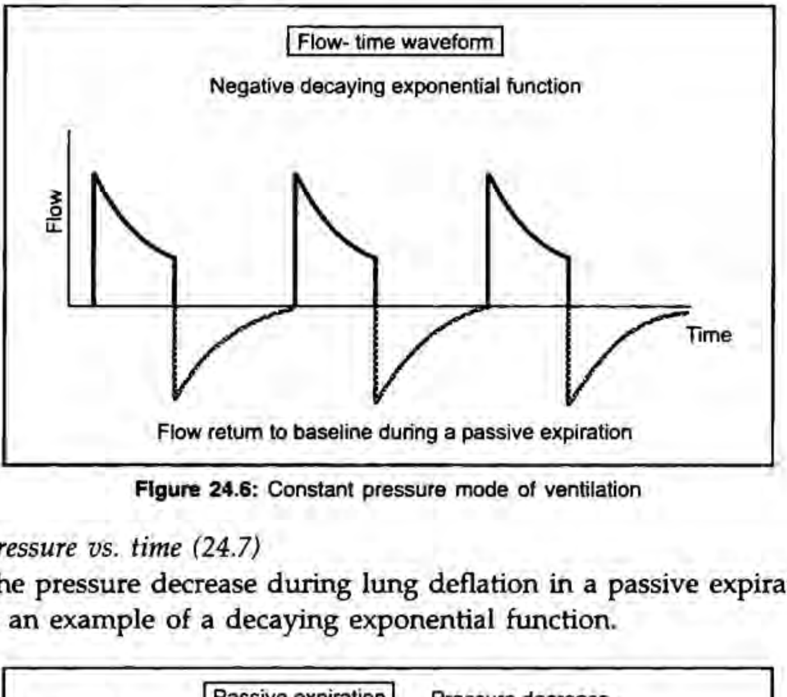


Figure 24.6: Constant pressure mode of ventilation

- *Pressure vs. time (24.7)*
  The pressure decrease during lung deflation in a passive expiration is an example of a decaying exponential function.

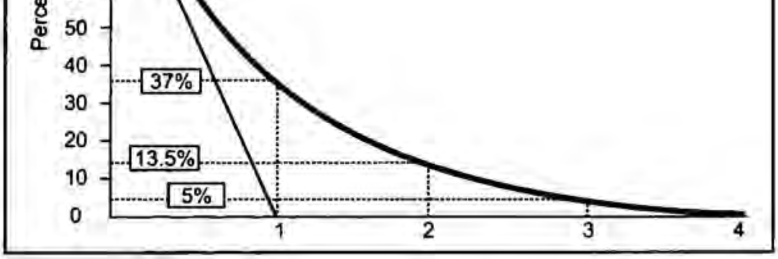


Figure 24.7: A decaying exponential function (The pressure decreases)

Figure 24.7—A decaying exponential function expresses the behaviour of a physical system where the rate of change of one variable is proportional to its magnitude only.

In this expression, as time progresses and the function y approaches zero, the rate of change of y decreases toward zero. Since the function y is always at its largest value at the beginning of the event (y0) its rate of change is also the largest at the beginning of the event and smallest at the end of the event.

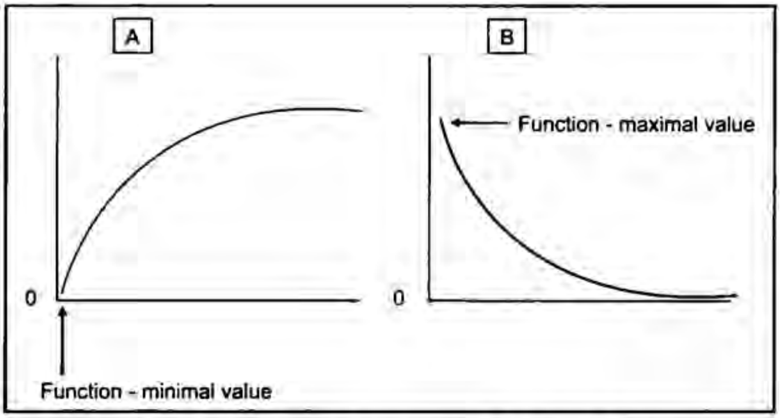
t – is the period of the time after onset of the event

y – is the value of the variable at time t

y0 – is the final value of y

In Figures 24.5 and 24.7, if the rate of rising or decaying were constant, a linear curve would reflect the behaviour of the function (thin lines). However, in exponential functions the rate of change is not constant over a period of time. The behaviour of the exponential functions is shown by the thick curve of figures.

In Figures 24.8A and 24.8B the evolution of the event is shown as a function of time.



Figures 24.8A and B: The evolution of the event is shown as a function of time at t = 0

At t = 0 the function is shown at its minimal value in Figure 24.8 A and at its maximal value in Figure 24.8B. After a period of time, both functions approach their respective final value. During the event, the rate of variation of an exponential function will depend on the value of the time constant of the system. Mathematically, after infinity of time constants, the value of the y-axis will never reach zero.

Percentage change in final or initial values for two types of exponential functions:

Time constants (τ)	*Rising exponential function(%) of final value*	*Decaying exponential unction (%) of initial value*
0	0	100
1	63.3	36.7
2	86.5	13.5
3	95.1	4.9
4	98.2	1.8
5	99.3	0.7

The table illustrates the relative value of five time constants in both, rising and decaying exponential function encountered in mechanical ventilation.

In a Rising Exponential Function, after

1. One time constant, the value of the variable on the y-axis increases to 63.3% of its final value.
2. Two time constants it increases to 95.1% of the final value.
3. Four time constants it increases to 98.2% of its final value.

In a Decaying Exponential Function, after

1. One time constant, the value of the variable on the y-axis decreases to 36.7% of its initial value (Fig, 24.9).

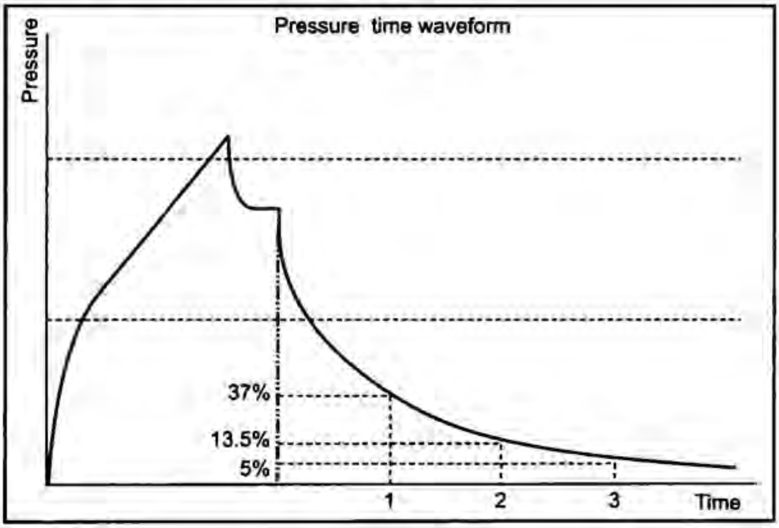


Figure 24.9: Decaying exponential function

2. Two time constants, the value decrease to 13.5 % of the initial value.
3. Three time constants the value decreases to 4.9 % of the initial value.
4. Four time constants, the value is only 1.8 % of the initial.

Effects of Variable Time Constants on Expiratory Volume (Fig. 24.10)

The emptying of lung occurs exponentially.

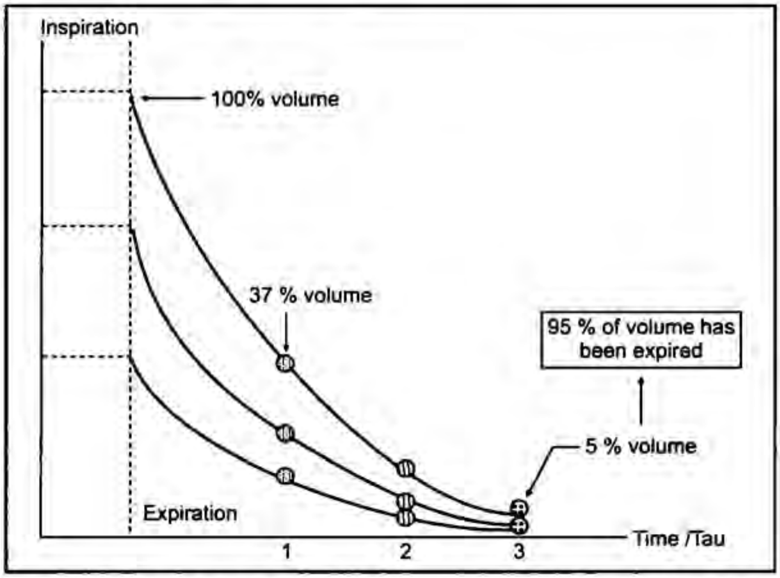


Figure 24.10: Effects of variable time constants on expiratory volume

It is observed that emptying process in units of the time constants shows that the lung volume has fallen from 100% to 37% within one time constant. After three time constants it has fallen to 5%, i.e. 95% of volume has been expired.

A Constant Pressure Mode of Ventilation—Flow-time Waveform (Fig. 24.11)

A – Exponential function with a short time constant
B – Exponential function with a long time constant

As its name implies, the time constants corresponds to a period of time.

A system with a short time constant will react very fast to a stimulus, as in–A. A system with a long time constant will react very slowly to a stimulus, as in – B. Both systems reach the same final value but over a different period of time

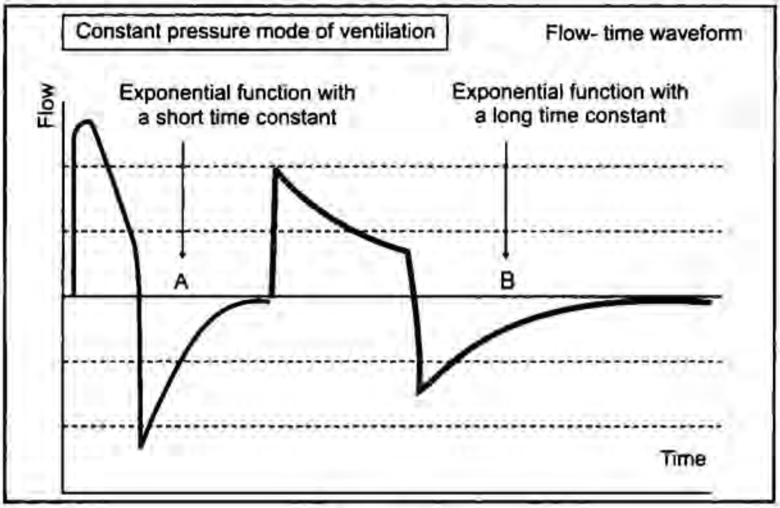


Figure 24.11

The actual value of one time constant is obtained through the product of compliance × resistance:

$$\tau = \text{Compliance} \times \text{Resistance}$$

As an example, a system with a total lung/thorax static compliance of 60 ml/cm/H_2O, an expiratory resistance of 0.13 cm H_2O per ml per sec and no auto-PEEP, has a time constant of—

$$\tau = \text{Compliance} \times \text{Resistance}$$

$$\tau = 60\ \text{ml/cm/H}_2\text{O} \times 0.13\ \text{cm H}_2\text{O/ml/sec.}$$

$$\tau = 0.78\ \text{seconds}$$

In mechanical ventilation, for practical purposes, an event is considered complete after three time constants. For the adult respiratory system the normal time constant is 0.78 seconds.

In a Decaying Exponential Function

1. A time constant of 0.78 seconds means that after one-time constants (0.78 sec.), the value of the variable on the y-axis decreases to 37% of its final value.
2. After two time constants (1.56 sec.) it decreases to 13.5% of its final value.
3. After three time constants (2.34 sec.) the value of the variable on the y-axis decreases to 5% of its final value.

In this example, if the expiratory time is shorter than 2.34 seconds (3 × 0.78), air trapping will be present, causing auto-PEEP. To prevent auto-PEEP and air trapping, the expiratory time should always be longer than 3 time constants.

Pressure-time Waveform From a Constant Flow Mode

Peak inspiratory pressure is 18 cm H_2O and end expiratory pressure is zero. Compliance is 83 ml/cm H_2O and resistance is 8 cm H_2O/l/sec.

$$t = \text{Compliance} \times \text{Resistance}$$

$$t = 83\ \text{ml/cm/}H_2O \times 0.082\ \text{cm}\ H_2O\text{/ml/sec}$$

$$t = 0.68\ \text{seconds}$$

The shape of expiration is a decaying exponential function. The product of compliance and resistance (0.68 sec) is the value of the time constant t for the entire system.

Summary of a Decaying Exponential Pressure-time Waveform

Time constant(t)	*Real time (Seconds)*	*Pressure (Cm H_2O)*	*% of initial pressure*
0	0.00	18.	100
1	0.68	6.48	36.7
2	1.36	2.43	13.5
3	2.04	0.882	4.9
4	2.72	0.324	1.8

Clinical Applications

1. During various modes of ventilation, the variable flow, pressure and volume will vary with time as an exponential function.
2. Depending on the mode of ventilation, compliance and resistance will affect both phases of each variable.
3. During inspiration, flow and pressure patterns are directly related to the ventilator (generator).
4. The clinician can control flow and pressure through ventilation strategies in order to optimize ventilation according to the compliance and resistance of the patient's respiratory system.

Flow-time Waveform where the Expiratory Time is Shorter than 3τ

The expiratory phase of the flow-time waveform is a negative phase decaying exponential function. Inspiration begins before the cycle completes three time constants, as a result air trapping occurs with resulting auto-PEEP (Fig. 24.12).

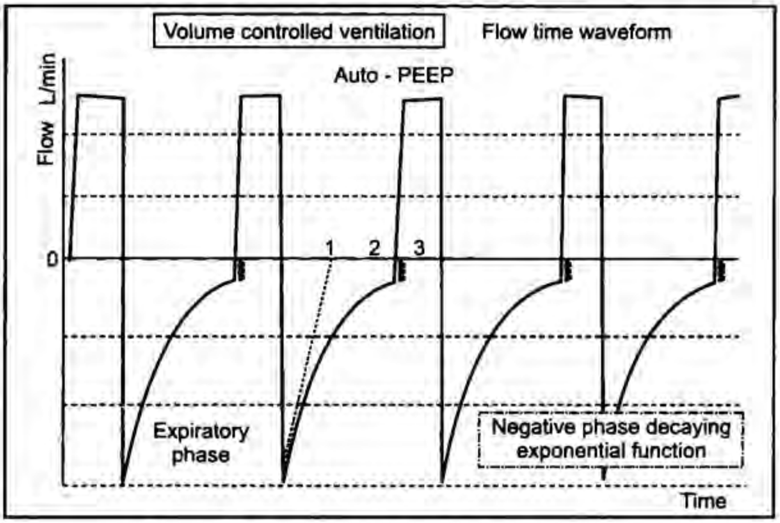


Figure 24.12: Expiratory time is shorter than 3τ

Clinical Applications

1. A flow-time waveform does not allow the clinician to quantify the amount of auto-PEEP produced by air trapping.
2. Auto-PEEP can be measured with the pressure-time waveform, using the end expiratory occlusion technique.
3. During expiration, flow, pressure and volume patterns cannot be directly manipulated by the clinician, except for determining the duration of each cycle when using control mode.
4. Passive expiration is then directly governed by elastic and resistive characteristics of the respiratory system.

Types of Time Constant

1. Short time constants – imply a fast rate of change and vice versa.
2. Long time constants – imply a slow rate of change.

Inspiratory and expiratory time constants are very close in normal value in a normal lung, but their differences may be accentuated in various disease states. In addition different segments of the lung may have different time constants.

Inspiratory Time

Thus to deliver volume to the lung, three to five time constants to be allowed during inspiration. (Normally 0.3 to 1.5 sec depending on the patient's age).

Expiratory Time

Likewise, if three to five time constants are not allowed in expiration, air trapping will occur, resulting in auto-PEEP. It takes a finite amount of time to inflate the lung. This factor is directly proportional to the compliance and the resistance. Passive inflation and deflation of the lung is normally exponential. Expiration is mostly passive due to the elastic recoil of the lung. This elastic recoil is attributable to:

1. Alveolar surface tension
    - Surface tension is greater at high lung volumes and lower at FRC.
2. Tissue elasticity

Since expiratory resistances are different, inspiratory and expiratory time is different. Typical values for resistance (pulmonary and apparatus) and compliance for an anaesthetized patient is compliance 50 ml/ cm H_2O and resistance 10 cm H_2O/lit/sec.

$$\begin{array}{lcl} \text{Compliance} \times \text{Resistance} & = & \text{Time constant} \\ 50\ \text{ml/cm}\ H_2O \times 0.01\ \text{cm}\ H_2O/\text{ml/sec} & = & 0.5\ \text{Seconds} \end{array}$$

During mechanical ventilation, for practical reasons, the value of the expiratory time constant reflects the characteristics of the global respiratory system.

1. In reality, the respiratory system consists of a multitude of zones, all with different time constants.
2. Various lung pathologies, like ARDS, consist of non-homogeneous lung zones with different compliance and resistance.
3. Diseased zones will have shorter time constants than normal zones with normal compliance.
4. Ventilatory strategies should aim at providing adequate ventilation according to the global time constant of the total respiratory system. It is commonly accepted that slowing end-inspiratory flow may allow better distribution of ventilation among lung units with different time constants.

Clinical Applications

1. Time constant is a measure of how quickly pressures in the proximal airways (or ventilator tubing) can be delivered or equilibrated in the alveoli
    - In one time constant 63% of the volume to be delivered (or emptied) is accomplished
    - 87% is reached by two-time constant
    - 95% in three time constants
2. It also reflects how quickly a lung can exhale after having been inflated.

Anaesthetic Application of Time Constant in Anesthesia Practice

1. An increase in resistance or decrease compliance gives rise to a longer time constant.
2. Different parts of the lung may have different time constants and this scatter of time constants is exaggerated in some forms of respiratory diseases such as asthma and emphysema.
3. A short inspiratory time will result in poor ventilation of those zones of the lung having a long time constant and there will be a resulting increase in V_A/Q mismatch.
4. Inspiratory and expiratory time constants may be determined because there are differences in airway caliber and lung compliance at different stages of the respiratory cycle. These are very close in value in a normal lung, but differ in various diseases.
5. A shortening of the time constant occurs when compliance or resistance or both are low. In ventilator treatment insufflations pressure will reach the alveoli very rapidly and the pressure drop on expiration will be very rapid.

Baby lung (Fig. 24.13)

It is important to emphasize that the measurement of both resistance and compliance only reflects the condition of the part of the lung that is ventilated. Thus a low compliance or a high resistance can be caused by small lung, i.e. the 'baby lung'.

In ARDS and other type of 'baby lung' as in severe asthma, most of the lung volume is trapped behind closed obstructed airways.

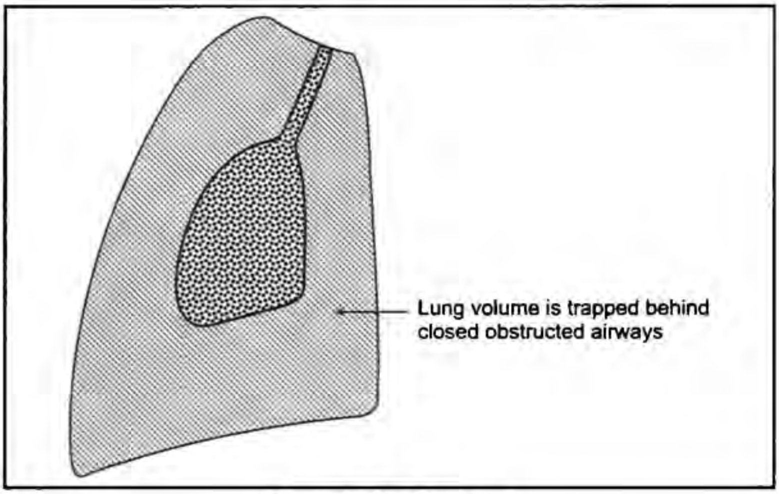


Figure 24.13: Baby lung

TIME CONSTANTS IN VARIOUS CONDITIONS

Infants with RDS

1. Infants with RDS have decreased compliance (stiff lungs) and Normal resistance.
2. In these patients, the time constant and corresponding time for pressure equilibrium will be shorter.
3. Short inspiratory and expiratory time may be appropriate during the period of peak severity of the disease but insufficient after recovery from RDS, when compliance is higher.

Infants with Diseases that Causes Increased Airway Resistance

Infants with diseases that cause increased airway resistance, such as MAS, asthma, Bronchopulmonary dysplasia (BPD) will have increased time constants due to slower filling and emptying of the alveoli.

Obese Patients or Patients with Kyphoscoliosis

The compliance of the chest wall contributes to the total compliance and unfortunately this is also related to the actual lung volume. This effect is even more marked if the chest wall compliance is low as seen in obese patients or patients with kyphoscoliosis.

CHAPTER

25

Monitoring of Lung Mechanics

IMPEDANCE TO LUNG INFLATION (FIG. 25.1)

Thoracic structures impede lung inflation. Therefore, a certain amount of force is required to overcome this impedance. A major impediment to lung inflation is elastic recoil of the lung and chest wall and the second major determinant of impedance to lung inflation is airway resistance.

The lungs and the thoracic cage are both elastic structures. They display a constant relationship between distending pressure and change in volume. The volume of air that moves into the lung is influenced by the alveolar pressure range and compliance. The alveolar pressure range is

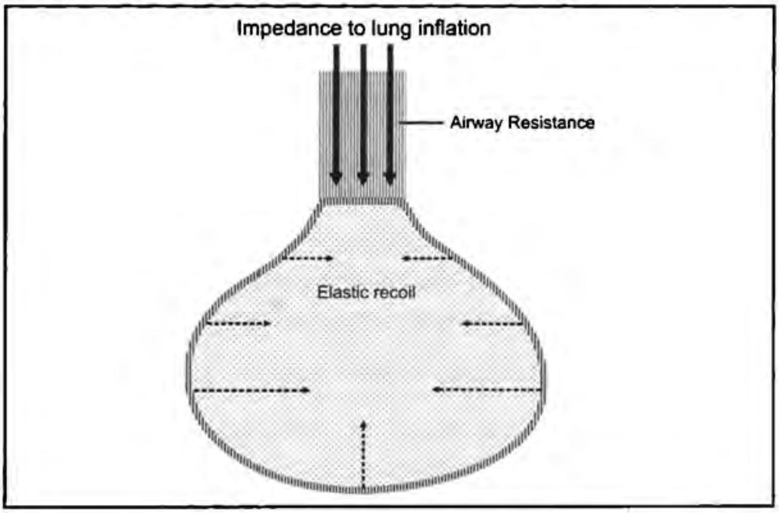


Figure 25.1: Impedance to lung inflation

the difference between the pressures in the alveoli at the end of inspiration to that of the pressure at the end of expiration.

1. *End inspiratory pressure*

   The end inspiratory pressure in the alveoli is primarily influenced by the pressure at the mouth end (atmospheric pressure when the patient is breathing spontaneously) and the pressure difference across the airways.

2. *End expiratory pressure*

   Similarly the end expiratory pressure in the alveoli is also influenced by the pressure at the mouth end (atmospheric pressure when the patient is breathing spontaneously) and the pressure difference across the airways.

This means pressure difference (transairway pressure) needed to move the required volume of air across the airways is determined by the resistance offered by the airways.

TOTAL COMPLIANCE (C_{TOT})

Total compliance has two components, lung compliance ($C_{pulm\ /\ Lung}$) and chest wall compliance (C_{CW}) and is normally 100 ml/cm H_2O. This compliance can directly influence the volume of air that can be moved into the lungs by muscular force. The compliance or distensibility of the lung and chest wall can be determined as the ratio of a change in lung volume (i.e. inflation volume) to a change in elastic recoil pressure (i.e. plateau pressure).

Flow/Volume Waveforms Produced by Volume Controlled Ventilation

The lungs are inflated at a constant flow rate and this produces linear increase in lung volume (Figs 25.2 and 25.3).

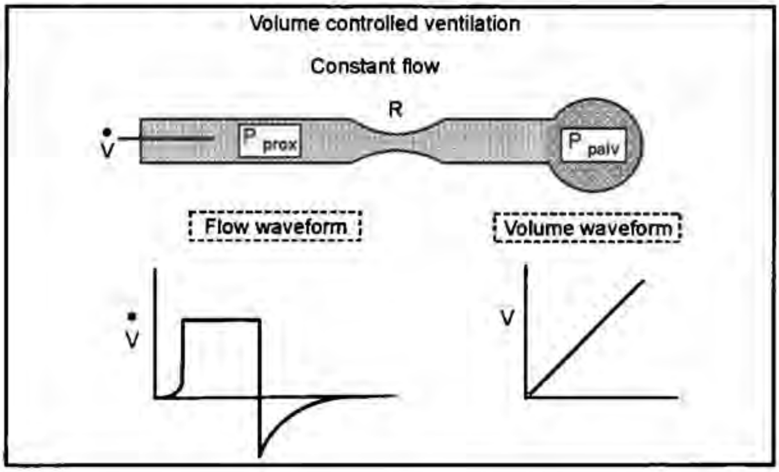


Figure 25.2: Flow/Volume waveform produced by constant flow (For colour version *see* Plate 11)

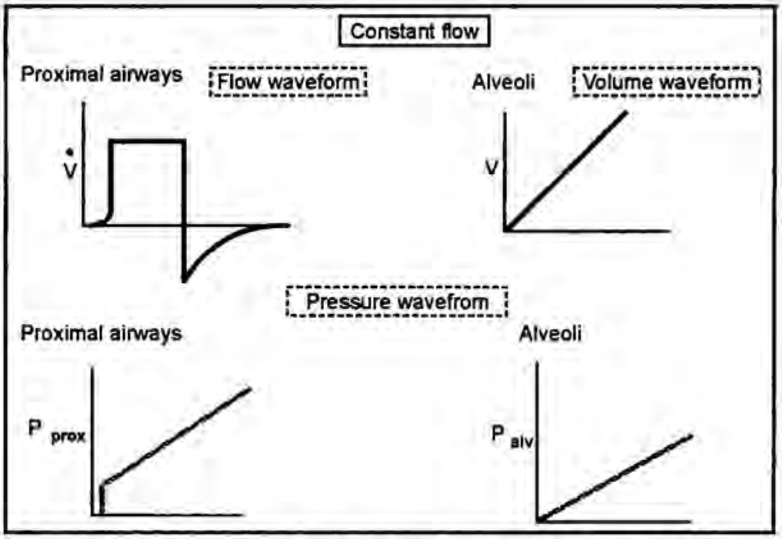


Figure 25.3: Flow/Pressure waveform produced by constant flow (For colour version *see* Plate 11)

Flow/Pressure waveforms produced by volume-controlled ventilation in normal and abnormal lung (Fig. 25.4)

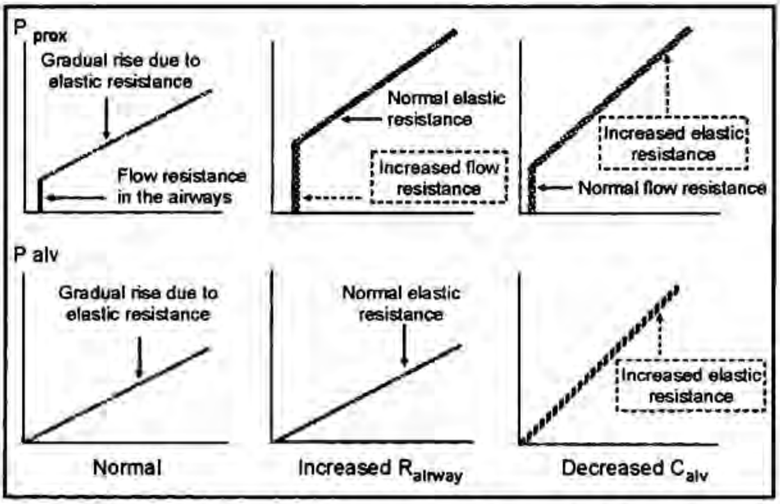

P prox
Gradual rise due to elastic resistance
Flow resistance in the airways
Normal elastic resistance
Increased flow resistance
Increased elastic resistance
Normal flow resistance
P alv
Gradual rise due to elastic resistance
Normal elastic resistance
Increased elastic resistance
Normal
Increased R airway
Decreased C alv

Figure 25.4: Flow/ Pressure waveforms produced by constant flow in normal and abnormal lung (For colour version *see* Plate 12)

- Lung with normal resistance and compliance
  - Proximal airways - The pressure in the proximal airways (P_{prox}) shows abrupt initial rise, followed by a more gradual rise through the remaining of lung inflation. The early, abrupt rise in proximal airway pressure is a reflection of flow resistance in the airways, followed by a gradual rise due to elastic resistance.

  - Alveoli - However the pressure in the alveoli (P_{alv}) shows only a gradual rise during lung inflation due to elastic resistance

- Lung with increased resistance and normal compliance:
  An increase in airway resistance magnifies the initial rise in proximal airway pressure, while the alveolar pressure at the end of lung inflation remains unchanged. Thus, when resistance in the airways increases, higher inflation pressures are needed to deliver the constant inflation volume, but the alveoli are not exposed to the higher inflation pressures.
- Lung with normal resistance and increased compliance:
  When the distensibility (compliance) decreases, the higher inflation pressures needed to deliver the inflation volume, this pressure is readily transmitted to the alveoli. The increase in alveolar pressure in noncompliant lungs to deliver constant volume, can lead to pressure-induced injury in compliant lung.

Monitoring Mechanical Properties

To avoid risk of lung injury observed with large inflation volumes in compliant lung, an alternative approach was introduced in recent years. The reduced lung volumes are recommended (5 -10 ml/kg), which helps not only to reduce injuries in compliant lung, but also helps to improve recovery.

During the spontaneous breathing, the mechanical properties of the lungs (i.e. elastic recoil of the lungs and resistance to flow in the airways) can be monitored with pulmonary function tests. However, during mechanical ventilation the proximal airway pressure can be used to assess pulmonary function.

PROXIMAL AIRWAY PRESSURE

Positive-pressure mechanical ventilators have a pressure gauge that monitors the proximal airway pressure during each respiratory cycle. The components of this pressure are Peak pressure [PIP/PAP/P_{peak}] and Plateau pressure ($P_{plateau}$).

Peak Inspiratory Pressure (PIP) (Fig. 25.5)

PIP represents the peak dynamic pressure, which is used to deliver the tidal volume by overcoming non-elastic (airways) and elastic (lung parenchyma and chest wall) resistance. The peak inspiratory pressure is a function of:

- Inflation volume
- Flow resistance in the airways
- Elastic recoil force of the lungs and chest wall

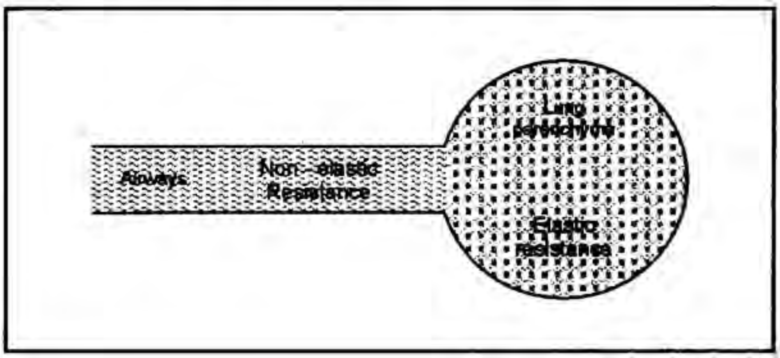


Figure 25.5: Peak inspiratory Pressure

At a constant inflation volume, the peak inspiratory pressure is directly related to airflow resistance or to the elastic recoil force (elastance) of the lungs and the chest wall or both. Therefore, when the inflation volume is constant, an increase in peak inspiratory pressure indicates an increase in either airway resistance or elastance (inverse of compliance) of the lungs and chest wall, or both.

Plateau Pressure - $P_{plateau}$ (Fig. 25.6)

Plateau pressure is the pressure needed to maintain lung inflation in the absence of airflow and it will indicate elastic (lung and the chest wall parenchyma) resistance and is often referred to as a close estimate of the alveolar pressure.

The differentiation of both pressures is done by inspiratory hold or occluding the expiratory port at the end of inspiration. When the inflation

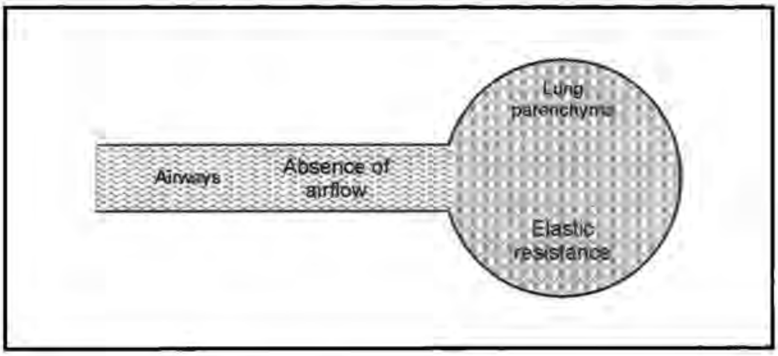


Figure 25.6: Plateau pressure

volume is held constant in the lungs, occluding the tracheal tube or ventilator tubing to prevent lung deflation, the proximal airway pressure decreases initially and then reaches a steady level. This steady pressure is called the end-inspiratory plateau pressure.

Because no airflow is present when the plateau pressure is created, the pressure is not a function of flow resistance in the airways, but directly proportional to the elastance of the lungs and chest walls.

PROXIMAL AIRWAY PRESSURES AT THE END OF POSITIVE-PRESSURE LUNG INFLATION SHOWING PEAK AND PLATEAU PRESSURE (FIG. 25.7)

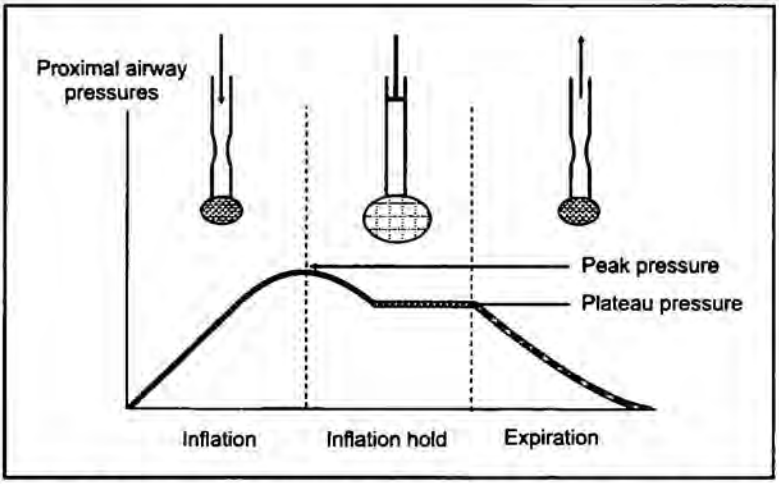


Figure 25.7: Proximal airway pressures at the end of positive-pressure lung inflation (For color version *see* Plate 12)

Measurement of Compliance

Total static compliance (Cst_{tot}) or Static compliance (C_{stat})

Static characteristics thus reflect the elastic properties of the respiratory system. Because the proximal airway pressures are transthoracic pressures (i.e. measured relative to atmospheric pressure) and not transpleural pressures (i.e. measured relative to intrapleural pressure), the compliance measurement includes the chest wall as well as the lungs. Total static compliance describes the delivered tidal volume relative to the airway pressure required to overcome the elastic forces of the respiratory system under static (zero flow) condition.

Normal values of static compliance are 40-60 ml/cm H_2O. It is lower in intubated patients, depending on the internal diameter of the ET tube. As a rule of thumb normal values will be 1 ml/cm H_2O/kg of body weight (lean body mass). Newborn 5–6 ml/cm H_2O.

Cst_{tot} is expressed by the following equation:

$$\text{Static compliance} = \frac{\text{Volume change in liters}}{\text{Pressure change in cm of } H_2O}$$

$$= \frac{V_T \text{ (corrected for compressible tubing volume)}}{P_{plateau} - PEEP_{total} \text{ (auto-PEEP + PEEP)}}$$

SHIFTING OF THE DYNAMIC AND STATIC COMPLIANCE CURVES (FIG. 25.8)

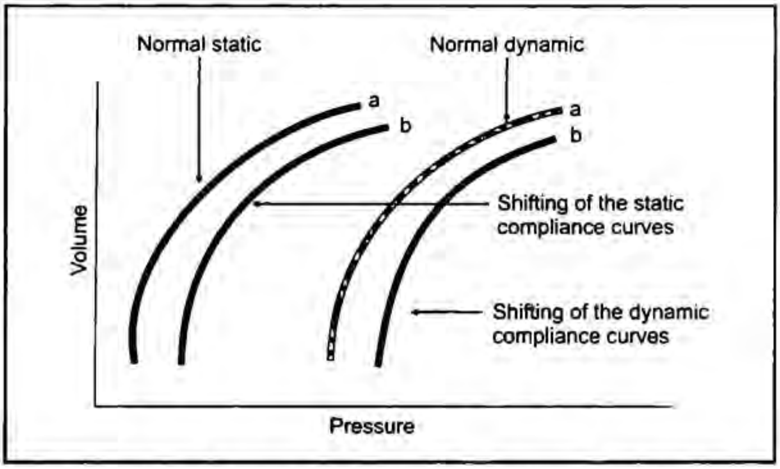

Normal static
Normal dynamic
a
b
a
b
Volume
Shifting of the static compliance curves
Shifting of the dynamic compliance curves
Pressure

Figure 25.8: Shifting of the dynamic and static compliance curves

Both curves shift to the right with a decrease in static compliance. Note that the static compliance curve and the dynamic compliance curve are both shifted to the right, [Static (b) and dynamic (b)].

Improvement in compliance to its normal state will cause both compliance curves to return to their original positions [static (a) and dynamic (a)].

Clinical Significance of Static Compliance

Changes in static compliance are associated with changes in lung elasticity; lung pathology that increases lung recoil and decreases lung volume will decrease the static compliance.

- This parameter can give important information on selection of an adequate PEEP and tidal volume.
- Change in static compliance gives important information, e.g. atelectasis, pneumothorax, ARDS, retained secretions.

- During mechanical ventilation the respiratory system will demonstrate a low static compliance both at low and high lung volumes. The midrange best working zone can be defined and used during setting of the ventilator.

Dynamic Compliance

Dynamic characteristics thus reflect the resistive and elastic properties of the respiratory system. Dynamic compliance is the compliance measured during spontaneous breathing, reflecting normal elastic recoil of the lungs.

Total dynamic compliance describes the delivered tidal volume relative to the peak airway pressure required to overcome the resistive and elastic forces of the respiratory system under dynamic conditions. Normal values of dynamic compliance are 30-40 ml/cm H_2O. Dynamic characteristics values are 10-20% lower than static compliance.

This is expressed by the following equation:

$$\text{Dynamic compliance} = \frac{V_T \text{ (corrected for compressible tubing volume)}}{\text{PIP-PEEP}_{\text{total}} \text{ (auto-PEEP + PEEP)}}$$

SHIFTING OF THE DYNAMIC COMPLIANCE CURVE (FIG. 25.9)

The dynamic compliance curve shifts to the right with a decrease in dynamic compliance (increase in nonelastic or airway resistance). Note that shifting of the dynamic compliance curve occurs independently [dynamic (b)] and that the position of the static compliance curve is unchanged.

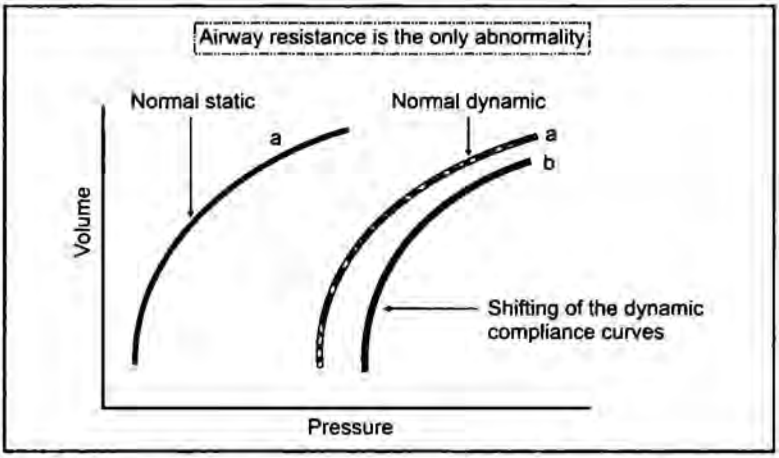


Figure 25.9: Shifting of the dynamic compliance curve

Improvement in nonelastic or airway resistance to its normal state will cause the dynamic compliance curve to return to its original position [dynamic (a)].

Clinical Significance of Change in Dynamic Compliance

The difference between static compliance and dynamic characteristics can be used as an indirect index of flow resistive properties of the respiratory system.

- In conditions where airway resistance is the only abnormality, dynamic compliance can change independently without a corresponding change in static compliance. Bronchospasm can decrease dynamic compliance while leaving static compliance unaffected. When bronchospasm is resolved, dynamic compliance returns to normal.
- Endotracheal tube size is an important element in gas flow through the breathing circuit, affecting resistance and dynamic characteristics of the respiratory system.
- Any conditions causing changes in static compliance cause similar changes in dynamic compliance. In patients with atelectasis, airway obstructions, kinking of endotracheal tube both static and dynamic compliance measurements are decreased simultaneously. When atelectasis is resolved, both compliance measurements are increased and returned to normal concurrently.

The Following Factors Influence the Compliance Measurement

- *PEEP*

  PEEP increases the plateau/PIP pressure. Therefore, the level of PEEP (either externally applied and/or auto-PEEP) should be subtracted from the plateau/PIP for the compliance determination.
- *Compressible volume*

  The connector tubing between the ventilator and patient expands during positive-pressure lung inflations and the volume is lost in this expansion reducing the inflation volume reaching the patient. Because of this discrepancy, the predetermined volume setting on the ventilator should not be used as the inflation volume for the compliance calculation. Instead, the exhaled volume, which is usually displayed on the ventilator panel, should be used.
- *Measurement is only during passive ventilation*

  Contraction of the chest wall muscles can reduce the compliance determination. It is mandatory that compliance measurement should be performed only during passive ventilation. However during passive ventilation, the chest wall can account for 35% of the total thoracic compliance.

Compliance measurements should be done frequently, so that a trend can be established. Interpretation is of little value with a single compliance measurement. Patient's spontaneous respiratory efforts can have problems both in decreasing pleural and airway pressure during inspiration or during expiration, thereby measuring the true distending pressure.

If no change in peak pressure occurs, it does not necessarily mean that there has been no change in lung mechanics. The conclusion is that measurements of compliance and resistance will be more or less useless in the spontaneously breathing patient as well as in supported ventilation modes.

The sensitivity of the proximal airway pressures in detecting changes in lung mechanics is unknown. The pressures are used to evaluate an acute deterioration in respiratory status.

ACUTE RESPIRATORY DETERIORATION (FIG. 25.10)

1. If the peak pressure is increased but plateau pressure is unchanged – it denotes an increase in airway resistance
   - *Obstruction of tracheal tube or airway obstruction from secretions* Airways suctioning is indicated to clear secretions

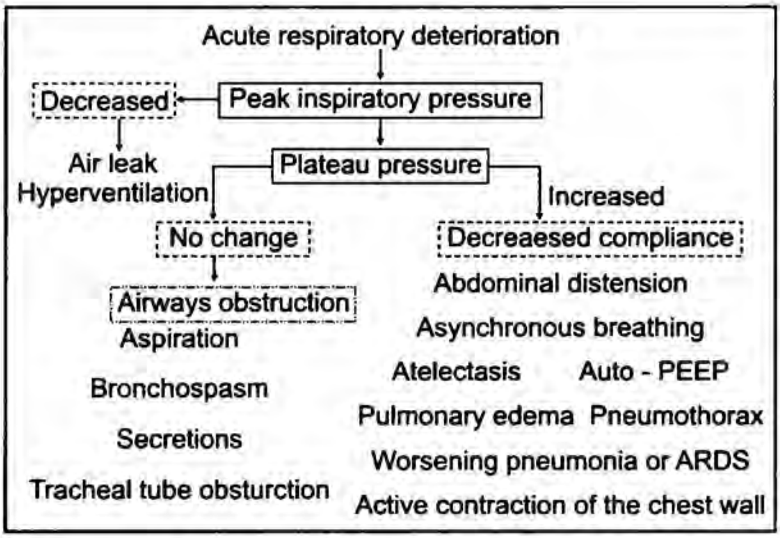


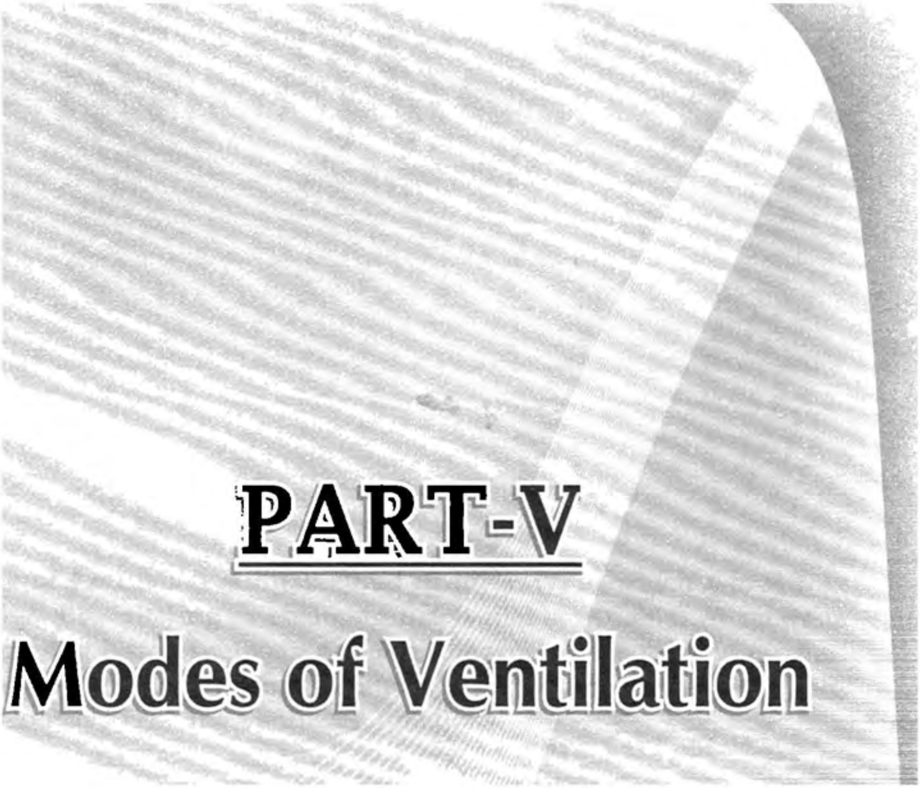
Figure 25.10: Acute respiratory deterioration

- *Acute bronchospasm*

  An aerosolized bronchodilator is treatment needed. Ventilator dependent patients often receive aerosolized bronchodilator treatments routinely, without documented need or benefit. If an aerosol bronchodilator treatment does not produce a decrease in peak inspiration pressures (indicating bronchodilation) there is little justification for continuing aerosol treatments.

2. Peak and plateau pressures are both increased– it denotes decrease in distensibility of the lungs and chest walls
   - Pneumothorax, lobar atelectasis, acute pulmonary edema
   - Worsening pneumonia or ARDS
   - Active contraction of the chest wall
   - Increased abdominal pressure
   - Patients with obstructive lung disease who became tachypneic can develop auto-PEEP.
3. If the peak pressure is decreased– it denotes an air leak in the system
   - Tubing disconnection
   - Cuff leak

   The lung should be manually inflated with an Ambu bag and cuff leak should be listened for during lung inflation.



PART-V

Modes of Ventilation

CHAPTER

26

What is Mode?

Mode of ventilation is the method or the way, in which a breath is delivered by altering or changing the available variables. A mode is nothing but how a ventilator performs the work of respiratory muscles. A ventilator mode can also be defined as a set of operating characteristics that controls how the ventilator functions.

An Operating Mode can be Described Depending On

- What triggers the ventilator into inspiration and cycle into expiration
- What variable limits during inspiration – what sustains inspiration
- Whether or not the mode allows only mandatory breaths, spontaneous breaths or both

To understand the terminology it is mandatory to know the components of a mode

COMPONENTS OF A MODE

1. Type of breath
    - Mandatory breath
    - Assist breath
    - Support breath
    - Spontaneous breath
2. Control variables
    - Pressure control
    - Volume control
    - Dual control

3. Phase variables
    - Trigger variable
    - Limiting variable
    - Cycling variable
    - Baseline variable
4. Conditional variables

Types of Breaths

1. A mandatory or machine breath (Fig. 26.1)
A mandatory breath is a breath started and ended by the machine.

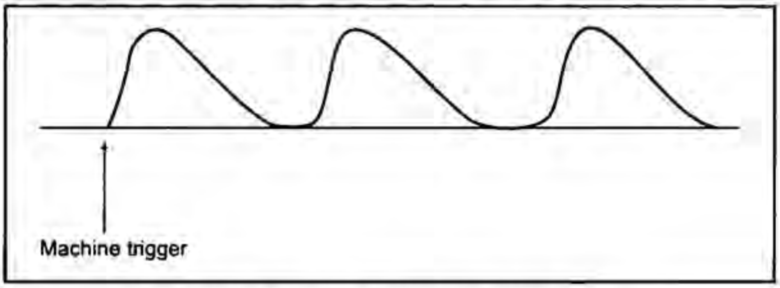


Figure 26.1: Mandatory or Control or Machine breath

2. Assisted control breath (Fig. 26.2)
Assisted breath is started by the patient but ended by the machine.

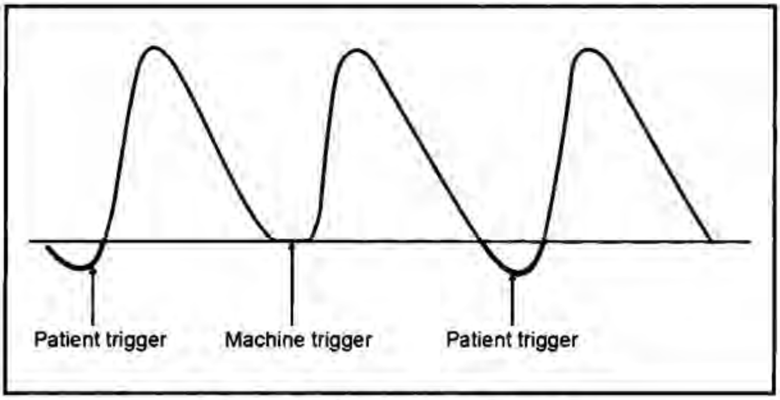


Figure 26.2: Assisted control breath

3. A spontaneous breath (Fig. 26.3)
A spontaneous breath is a breath started and ended by the patient.

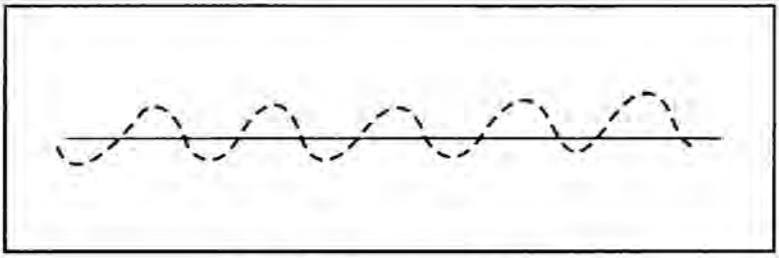


Fig. 26.3: A spontaneous breath

4. Support breath (Fig. 26.4)
   A support is a type of spontaneous breath, where inspiratory pressure is more than the base line.

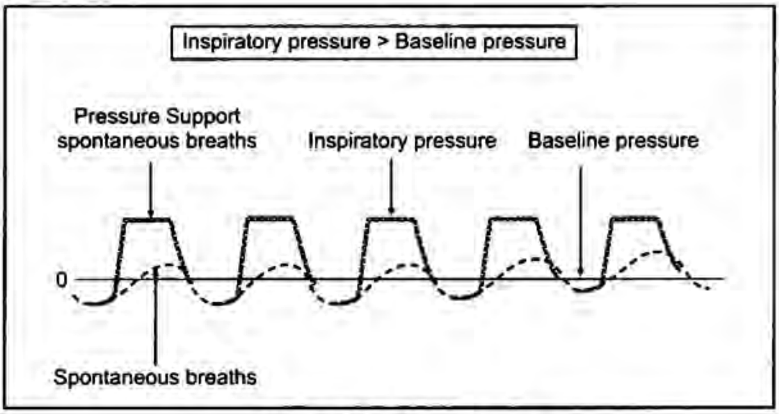

Inspiratory pressure > Baseline pressure
Pressure Support spontaneous breaths
Inspiratory pressure
Baseline pressure
0
Spontaneous breaths

Figure 26.4: Support breath

Control Variable

1. Pressure control
   Pressure control simply means the breaths are pressure constant and volume variable.
2. Volume control
   Volume control simply means the breaths are volume constant and pressure variable.
3. Dual control mode
   Dual control modes are newer modes which are capable of switching from one to another, e.g. pressure regulated volume control (PRVC), Auto mode, Adaptive pressure ventilation (APV).

Phase Variables (Fig. 26.5)

The variable that examined during a particular phase (inspiration and expiration), is termed a phase variable. The understanding of phase variable is useful for examining how a ventilator starts, sustains and ends an inspiration. Each breath either spontaneously or mandatory goes through four phases.

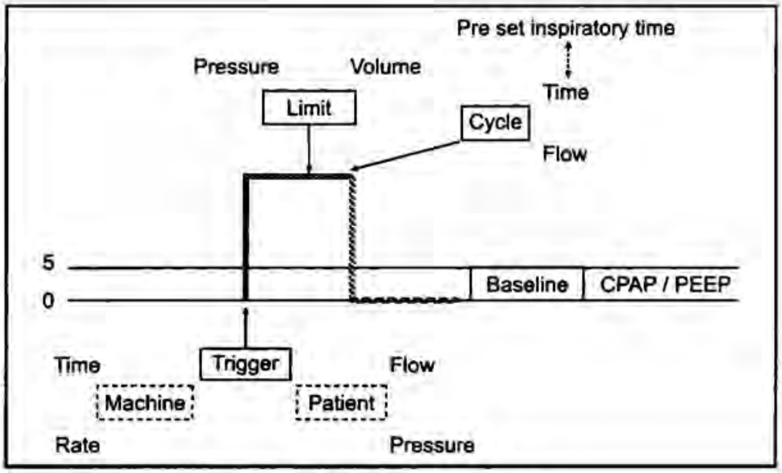


Figure 26.5: Phase variables

Trigger Variable – What starts inspiration

- *Machine trigger or time trigger:*
  Time is a trigger variable when the ventilator starts a breath according to a set frequency. Breath is initiated when a preset time interval for one complete respiratory cycle (inspiratory time and expiratory time) is complete.
- *Patient trigger:*
  Pressure, flow measured by the ventilator and used as a variable to initiate inspiration

  Pressure trigger is when the ventilator senses a drop in baseline pressure when caused by the patient's inspiratory effort and triggers a breath.

  Flow trigger when the ventilator senses the patient's inspiratory effort in the form of flow.

Limit Variable – What sustains inspiration

Limit variable means available variable is not allowed to rise above a preset value during the inspiratory time.

- Pressure limit
- Volume limit

Cycling Variable – what ends inspiration

The variable that is measured and used to end inspiration is called cycle variable.

- Time cycled – cycling is due to set inspiratory time
- Volume cycled
- Flow cycled

Baseline Variable – variable during expiration

The variable that is controlled during the expiratory phase or expiratory time, e.g. CPAP/PEEP

Conditional variable

Conditional variables are defined as patterns of variables that are controlled by the ventilator during the ventilator cycle. For each breath the ventilator creates a specific pattern and phase variables. The ventilator may either keep this pattern constant for each breath or it may introduce other patterns, e.g. one specific pattern for mandatory and another pattern for spontaneous breath (SIMV).

Accordingly Type of a Breath, Ventilation may be Labeled as a

1. *Controlled Mechanical Ventilation*
   When the ventilator delivers a tidal breath irrespective of the patient's effort
   - Pressure-control breaths are pressure constant
   - Volume-control breaths are volume constant
2. *Assist Control Ventilation*
   When the ventilator delivers a tidal breath sensing the patient's effort, i.e. patient can start (trigger) the breath.
   - Assisted pressure control
   - Assisted volume control
3. *Dual Control Ventilation*
   In this mode there is advantage of both volume and pressure control ventilation

4. *Support Ventilation*

   Patient initiates a breath in which ventilator supports with a constant pressure or volume
   - Pressure support
   - Volume support

The Common Modes of Ventilation are (Fig. 26.6)

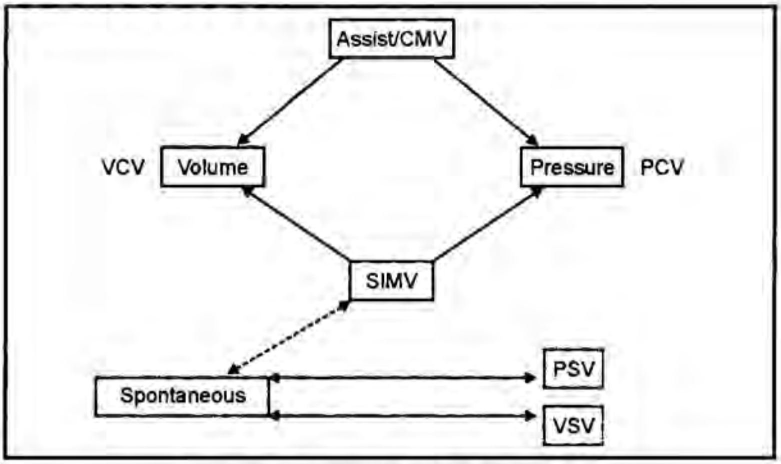


Figure 26.6: Common modes of ventilation

CHAPTER

27

Controlled Mandatory Ventilation

Controlled mandatory ventilation (CVM) /Assist mandatory ventilation are full support modes. Full support modes are required in acutely ill patients who require guaranteed and often high minute ventilation. These modes perform most or all of the work of respiratory muscles, necessary to maintain adequate minute ventilation. Because of full support, these modes also reduce the oxygen and energy consumed by the respiratory muscles.

Controlled mandatory ventilation is mechanically the simplest mode and therefore the most mechanically reliable mode of ventilation. In controlled mandatory ventilation, inspiration is introduced automatically and independently of spontaneous breathing, therefore there is no synchronization unless the patient is sedated or paralysed.

CMV can be further named as:

- IPPV – Intermittent positive pressure ventilation
  If the PEEP is zero the type of ventilation is called IPPV
- CPPV – Continuous positive pressure ventilation
  If the PEEP is greater than zero the type of ventilation is called CPPV.

DESCRIPTION OF CMV (FIG. 27.1)

Controlled mandatory ventilation is a mode of ventilation in which all breaths are mandatory or controlled. All breaths are delivered by the ventilator (ventilator controls) at a preset frequency, preset tidal volume or pressure and preset inspiratory time. The frequency at which triggering takes place is determined by the back up rate (preset frequency) set on the ventilator.

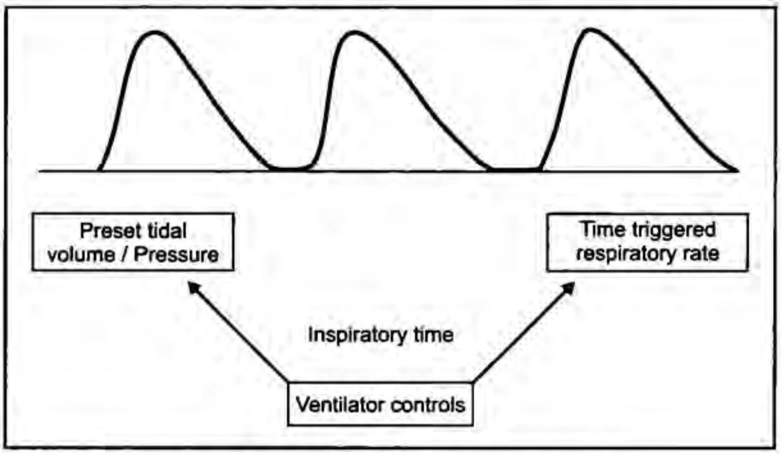

Preset tidal volume / Pressure
Time triggered respiratory rate
Inspiratory time
Ventilator controls

Figure 27.1: Mandatory or control breath

COMPONENTS OF CMV MODE (FIG. 27.2)

1. Types of breath - All breaths are mandatory breaths. Each breath delivers a mechanical tidal volume
2. Trigger – Time (preset frequency or backup rate). Every breath in the control mode is time triggered.
3. Limit/control – Volume or pressure
4. Cycle – Inspiration is terminated by the delivery of a preset of a tidal volume (volume cycled) or preset inspiratory time (time cycle).

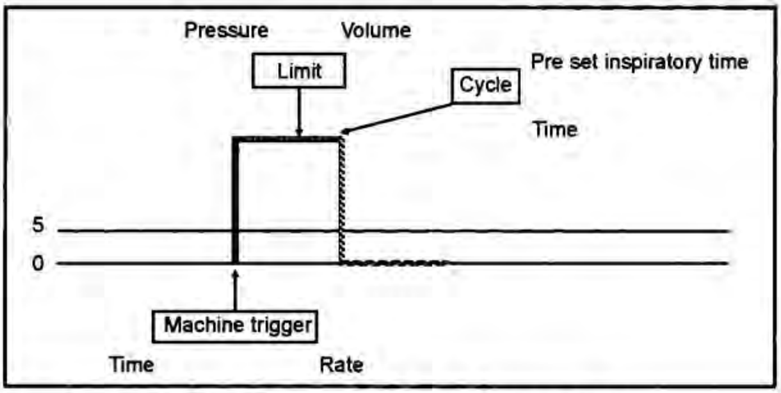


Figure 27.2: Components of mandatory breath

CLINICAL APPLICATIONS OF CMV/AMV

1. In the control mode, a patient cannot change the ventilator rate or breathe spontaneously.
2. The ventilator controls the total breathing work of inspiration and also the size of each given tidal volume

3. Since the ventilator controls the patient's tidal volume and respiratory rate, the ventilator controls the patient's minute volume and $PaCO_2$.
4. The control mode should only be used when the patient is properly medicated with a combination of sedatives, respiratory depressants and/or neuromuscular blockers.

PRECAUTIONS

If the patient attempts to ventilate spontaneously in this mode, he becomes dysphoric. These patients can neither trigger breath nor inspire air through the ventilatory circuit. Any spontaneous inspiratory effort is just like attempting to inspire through a completely obstructed airway. Regardless of how vigorous the patient's effort is, no gas flow would be delivered to the patient until the ventilator automatically becomes time triggered. It is most likely to be psychologically devastating for the patient to realize that he or she has no control over his breathing requirements. All measures should be taken to prevent agitation or dysphoria. The patient's respiratory effort should be suppressed by sedative, intentional hyperventilation or muscle relaxants.

INDICATIONS OF CONTROL MANDATORY VENTILATION

When the patient is first intubated, assist/control is commonly used because it provides maximum ventilator assistance. It also guarantees lower limit of delivered ventilation.

1. *To provide maximum ventilatory support:*
   CMV will provide maximum ventilatory support to a patient with marginal cardiorespiratory reserve. Full support improves gas exchange, or cardiac function.
2. *Controlling minute ventilation:*
   During hyperventilation minute ventilation has to be controlled, especially when reduction in cerebral blood volume and intracranial pressure are urgent priorities, after closed head injury.
3. *Reducing oxygen consumption:*
   Patients with respiratory muscle fatigue with a poor cardiac output, during periods of cardiovascular instability, patients with high neuromuscular drive or unstable respiratory drive, will need complete and maximal rest. In these patients it is advantageous to reduce oxygen consumption by giving complete rest to the respiratory muscles. The minimum period of rest especially after cardiac arrest is 12- 24 hours. Reducing oxygen consumption in a patient with critical coronary ischemia such as circulatory shock or acute pulmonary oedema may prove life saving. Suppressing ventilatory effort can

improve arterial oxygenation and lung volume recruitment in agitated ARDS patients.

4. *Ventilatory total control facilitates the therapeutic application of nonphysiologic breathing patterns.*
   Intervention such as inverse ratio ventilation, permissive hypercapnia requires silencing patient effort, CMV facilities the therapeutic application of these nonphysiologic breathing patterns due to total support.
5. *To reduce agitation:*
   Fighting" or "bucking" the ventilator often means that the patient is severely distressed and vigorously struggling for breathe. Patient fights the ventilator in the initial stages of mechanical ventilator support; their rapid spontaneous inspiratory effort becomes asynchronous with the ventilator's ability to provide an adequate inspiratory flow. The typical result is that the patient will be attempting active exhalation while the ventilator is delivering a breath. This results in high-pressure limit cycling which decreases the ventilator delivered tidal volume. CMV will reduce agitation by suppressing spontaneous effort.
6. *Helps to heal the injured chest wall:*
   Patients with a crushed chest injury, in which spontaneous inspiratory efforts produce significant paradoxical chest wall movements, may delay healing.
7. *Tetanus or status epilepticus or seizure activities:*
   Tetanus or seizure activities interrupts the delivery of mechanical ventilation hence CMV is strongly recommended.
8. *To facilitate the monitoring of respiratory mechanics:*
   It is rarely a sufficient reason to give deep sedation or pharmacologic paralysis to monitor respiratory mechanics.

COMPLICATIONS ASSOCIATED WITH THE CONTROL MODE

Since the patient's spontaneous respiratory drive will have been blunted with sedation/paralysis in the control mode, the patient is totally dependent on the ventilator for ventilation and oxygenation.

1. *Hypoxia during accidental disconnection:*
   The primary hazard associated with the control mode is potential for apnea and hypoxia if the patient gets accidentally disconnected from the ventilator.
2. *Respiratory alkalosis:*
   CMV is unresponsive to changing minute ventilation requirement of the patient, hence appropriate adjustment should be made time to time to avoid acid base disturbances.

3. *Respiratory muscle atrophy:*
   CMV is total mechanical support of ventilation which inhibits contraction of respiratory muscle. Patients are prone to respiratory muscle atrophy due to disuse.

MANDATORY VENTILATORY ALARMS

Volume and pressure alarms are complimentary to each other in any mode. Lower limit of alarms is essential to safe guard safety of patients with CMV due to accidental disconnection

1. Low exhaled volume alarm
2. Low inspiratory pressure alarm

USE OF SEDATION IN CMV

High respiratory rate results in intrinsic PEEP or wasted ventilation. Therefore sedation is always needed to establish ventilatory control in the conscious or semiconscious patient with moderate to high minute ventilation requirements. Use of sedation to improve comfort or inhibit excessive respiratory drive is a common practice. Muscle relaxants should be used sparingly because of their injurious potential and should not be used in conscious or lightly sedated patients.

USE OF MUSCLE RELAXANTS IN CMV

Precaution to be Taken when Using Muscle Relaxants

Severe diaphragmatic dysfunction occurs only after 2-3 days of controlled ventilation, independent of the use of paralyzing agent, with loss of more than 40% of power generation. It is likely that paralyzing agent potentiates or triggers diaphragmatic dysfunction by blocking protective mechanism against atrophy. Critical illness poluneuropathy, which frequently is associated with diaphragmatic dysfunction, increases the duration of mechanical ventilation and intensive care stay significantly. Deep sedation with short action is preferable to any form of paralysis. Paralytic drugs should not be used to totally suppress muscle activity but instead to weaken the force of the patient's effort.

Intermittent Use of Muscle Relaxants/Sedative

Another strategy is to use intermittent dosing, giving only as much as is needed to suppress excessive muscular activity. The periodic withdrawal of muscle relaxants also helps to decide adequacy of sedation. The technique which prevents total isolation of the muscle from neural impulses should be avoided. This is also true for sedative agents. Totally controlled ventilation may lead to muscle atrophy with or without paralysis.

CHAPTER

28

Assisted Controlled Mandatory Ventilation

Assist control ventilation is also called as Assisted mechanical ventilation (AMV), Assisted ventilation (AV), CMV with assist. In assist control ventilation the patient can initiate each mechanical breath (assisted ventilation), if this is not possible the ventilator provides machine breaths at a preselected rate (controlled ventilation). In assisted ventilation the ventilator provides mandatory breath, but the patient can trigger it. Therefore, the patient is ventilated synchronously with his spontaneous breathing effort.

Thus, assist control ventilation combines mandatory and assisted breaths that can be either volume controlled or pressure controlled. (Figs 28.1 and 28.2)

1. Mandatory breaths are delivered at set frequency
2. Between machine-initiated breaths (mandatory breaths), the patient can trigger the ventilator and receive assisted breaths at the volume or pressure set on the ventilator.
3. Mandatory and assisted breaths are delivered using the same limit and cycle variables.
   - Each control breath provides the patient with a pre-set, ventilator delivered tidal volume.
   - Each assist breath also results in a preset, ventilator delivered tidal volume, but does not allow the patient to take spontaneous breaths.
4. The only difference between CMV and A/C ventilation is that during A/C ventilation, the patient can also trigger a breath. With the A/C mode, the patient may increase the ventilator respiratory rate

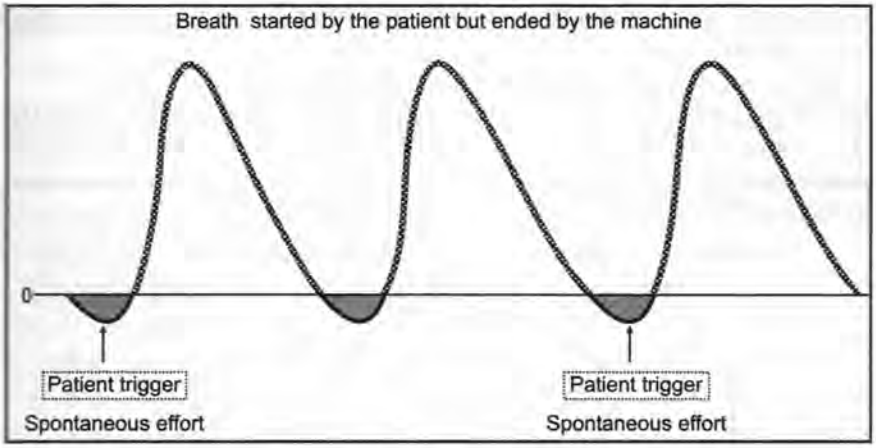


Figure 28.1: Assist control breath

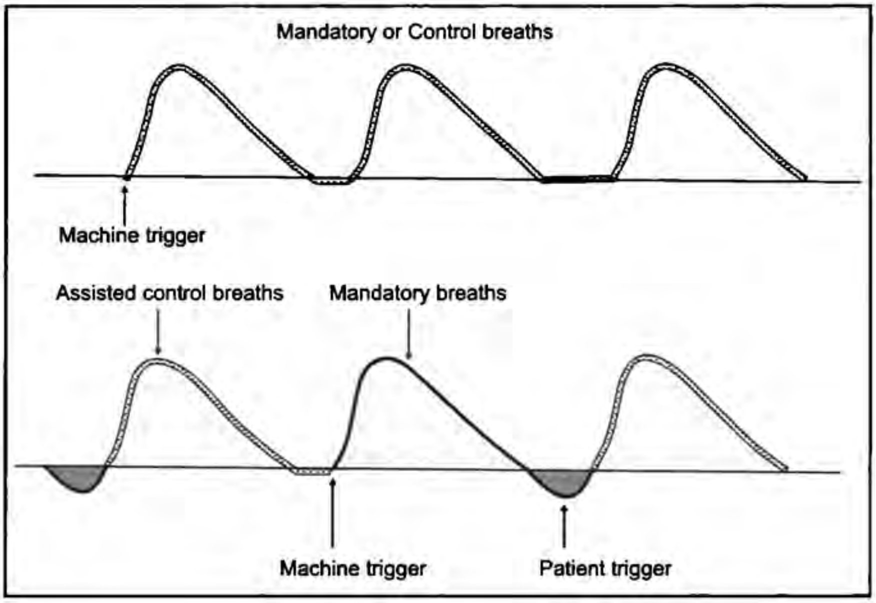


Figure 28.2: Mandatory and Assist control breath

(assist) in addition to the preset mechanical respiratory rate (control). In other words in ACV rate is always more than set or backup rate.

CLASSIFICATION OF ACV

1. Type of breath – Mandatory: Each breath, assist or control, delivers a preset mechanical tidal breath
2. Control – Pressure or volume

3. Trigger – Mechanical breaths may be either time triggered (control) or patient triggered (assist) (Fig. 28.3).
   - *Machine - time triggered* (based preset respiratory rate)
   - Patient triggered (based on patient effort and trigger sensitivity)

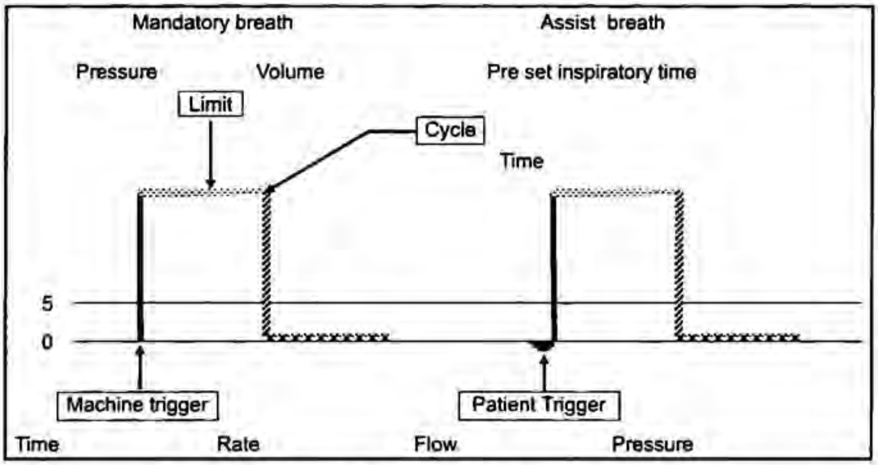


Figure 28.3: Trigger of Mandatory and Assist control breath

Trigger sensitivity: To get assisted breath patient must generate negative pressure or air flow in the airway sufficient to be sensed by the ventilator. If trigger sensitivity setting is too sensitive, then the patient will get breath with slightest movements. If sensitivity is set too low, then it requires the patient's increased effort and energy expenditure. It can also result in the patient being locked out, i.e. unable to trigger the ventilator to the point that no amount of patient effort can trigger the ventilator into inspiration.

4. Cycle - Machine: Inspiration is terminated either by the delivery of a preset tidal volume (volume cycled) or by the pressure limit (pressure cycled) or time cycle depending on settings.

CHANGES IN AIRWAY PRESSURE PRODUCED BY ACV

1. The tracing begins with a negative-pressure deflection produced by ACV, followed by a positive pressure machine breath.
2. The negative pressure represents a spontaneous inspiratory effort, which opens a pressure-activated valve that allows the machine breath to be delivered.

SAFETY FEATURE FOR ACV – BACKUP RATE

1. *Setting up backup rate less than patient's rate:*
   If the patient's respiratory rate falls below backup rate, the ventilator will automatically cycle at the backup rate. The Patient may not be able to trigger the ventilator due to sedative, unstable respiratory drive or respiratory muscle fatigue. A good strategy is to set the backup rate to provide 80% of the baseline minute ventilation.
   Clinical significance of backup rate
   - If the patient has stable assist rate of 12 breaths per minute, then the patient is triggering breaths every 5 seconds.
   - If the control rate is preset at 10 breaths per minute, the ventilator would deliver time-triggered breaths every 6 seconds.
   - However, since the interval between the assisted breaths is shorter than 6 seconds, no time –triggered breaths will be delivered.
   - If however, the patient's spontaneous breaths were to decrease less than the preset control rate, then the ventilator would begin delivering time-triggered breaths
2. *Setting up backup rate above patient's rate:*
   Patient's contribution can be decreased by sedation which suppress's the respiratory drive
   - Unstable cardiac output or low cardiac output
     With ACV oxygen consumption is decreased compared to spontaneous breathing. However, there is extra oxygen consumed by the respiratory muscles due to trigger compare to CMV, which can be a burden for the overtaxed cardiovascular system. In this type of a patient if the backup rate is set above the patient's rate it eliminates the respiratory muscle work or mode of ventilation can be changed the CMV.
   - Unstable respiratory drive
   - Patients with flail chest in whom vigorous inspiratory effort may interfere with healing.

ADVANTAGES OF THE AC MODE

- Spontaneous efforts during AMV can prevent or delay respiratory muscle atrophy.
- The patient work of breathing requirement in the AC is very small when the triggering sensitivity (pressure or flow) is set appropriately and the ventilator supplies an inspiratory flow that meets or exceeds the patient's inspiratory flow demand.

- If the patient's has an appropriate ventilatory drive, this mode allows the patient to control the respiratory rate and therefore the minute volume required to normalize the patient's $PaCO_2$.

DISADVANTAGES OF ASSIST CONTROLLED MANDATORY VENTILATION

Disadvantages of assist controlled mandatory ventilation are mainly due to increased frequency.

Severe Respiratory Alkalosis

If the patient is assisted at a high respiratory rate (i.e. > 20 to 25 breaths/minute) and the tidal volume is preset at 10-15 ml/kg, this will usually result in hypocapnia and respiratory alkalosis. Severe respiratory alkalosis primarily occurs in patients who are breathing rapidly, which leads to overventilation. If the patient's respiratory centre is either injured or diseased, the patient may have an inappropriately high respiratory drive leading to an excessive assist rate despite a low $PaCO_2$. The potential hazard is alveolar hyperventilation (respiratory alkalosis).

Though mechanical dead space may be used in this situation, it is generally considered safer to switch the patient to another mode of ventilation (e.g. SIMV) that limits the patient's ability to generate excessive minute volumes.

Auto PEEP

Increased respiratory rate decreases time for exhalation which results in hyperinflation. Auto-PEEP accompanies hyperinflation from inadequate lung emptying and can have the same adverse consequences as externally applied PEEP.

Fall of Cardiac Output

The mean intrathoracic pressure rises if the respiratory rate increases due to ACV. High mean intrathoracic pressure impairs venous return to the heart. Subsequent decrease in preload to the left ventricle, leads to rice fall in cardiac output.

INDICATIONS FOR ASSIST CONTROL MANDATORY VENTILATION (ACMV)

- The main reason for using ACV is to unload the inspiratory muscles and to improve gas exchange.
- Provide full ventilatory support for patients when they are first placed on mechanical ventilation.
- Same as CMV.

CHAPTER

29

Volume Controlled Ventilation

Volume control and pressure control are not modes; they indicate which variable is constant during breath delivery regardless of the changes in lung mechanics.

DEGREE OF FREEDOM

The degree of freedom includes those parameters of ventilation which are not directly influenced by the settings of the ventilator but depend on respiratory mechanics. In volume controlled ventilation the ventilation pressure represents the degree of freedom and in pressure controlled ventilation, the tidal volume represents the degree of freedom.

VOLUME CONTROLLED VENTILATION (VCV)

Volume controlled ventilation is actually flow controlled ventilation because the ventilator actually controls inspiratory flow. VCV is a volume controlled, time cycled mode which is completely controlled by the ventilator, with no participation by the patient. A preselected tidal volume is delivered due a constant flow.

PARAMETERS TO BE SET

- **Tidal volume**
- **Inspiratory flow rate**
- Machine rate
- A timing variable in the form of I: E ratio, the duty cycle (T_i/T_{tot}) or the inspiratory time
- Alarm limit

Tidal Volume

The selection of tidal volume in a given patient depends on the nature of the disease, the approximate minute ventilation requirements, pulmonary compliance, airway pressure, PaO_2, and $PaCO_2$. In general all patients can be started with a tidal volume of 10 ml/kg, stabilize the patient and then progressively reduce the tidal volume upto 5-8 ml/kg, so that the plateau pressure does not exceed 30 cmH_2O.

Patients with Normal Lungs

In patients with normal lungs, to start with set the tidal volume is set at 10 ml/kg, stabilize the patient and then progressively reduce the tidal volume to 5-8 ml/kg.

- Neuromuscular disease causing respiratory failure
- CNS disease, coma, poisoning
- Immediate postoperative period

In Patients whose Lungs are Stiff

In patients with stiff lungs, to start with, the tidal volume is set at 5-8 ml/kg.

- Acute lung injury
- Pneumonia

Clinical Significance of Low/High Tidal Volume

Risk with Low Tidal Volumes: Very low tidal volumes result in atelectasis, hypoventilation and hypoxemia.

Risk with High Tidal Volumes: Very high tidal volumes can lead to respiratory alkalosis, decrease in cardiac output by reducing venous return and predispose to barotrauma and volutrauma. There is definite evidence that overstretch of alveolar walls through very large tidal volume can induce lung injury.

Inspiratory Flow Rate

The inspiratory flow rate is usually set at 40-60 lit/min in volume targeted ventilation. The flow set at the ventilator must match the patient's peak inspiratory flow so that no negative pressure can occur. Flow rate can be increased up to 60-100 lit/min. in a patient with high inspiratory demands. Lower flow rates can be used to decrease peak inspiratory pressure in a patient of ARDS.

Machine/Respiratory Rate

The rate is generally set between 10-14 breaths min if the patient is clinically stable. In patients with stiff lungs, a higher rate may be required, so as to match the machine rate to the spontaneous breathing pattern of the patient. In COPD patients where minute ventilation needs to be restricted lower rates may be required. If the respiratory rate is too high, respiratory alkalosis, auto-PEEP and barotrauma can result. If too low, hypoventilation, hypoxaemia and patient discomfort are observed.

Inspiratory:Expiratory Ratio

The I:E ratio to start with is set at 1:2 or 1:3 to allow sufficient time for expiration.

Minute Ventilation

Minute ventilation is set according to the approximate ventilatory requirements of the patient. Initial minute ventilation can be decided around 5-10 lit/min; however it is also influenced by the nature of the disease and the altered respiratory mechanics, for which ventilatory support is required.

Output Variables of VCV - Airway Pressure (Fig. 29.1)

The airway pressure (ventilatory pressure) is dependent on the set tidal volume, inspiration time, directly proportional to the airway resistance and indirectly proportional to the compliance. An increase in the resistance and decrease in compliance will lead to an increased airway pressure and vice versa.

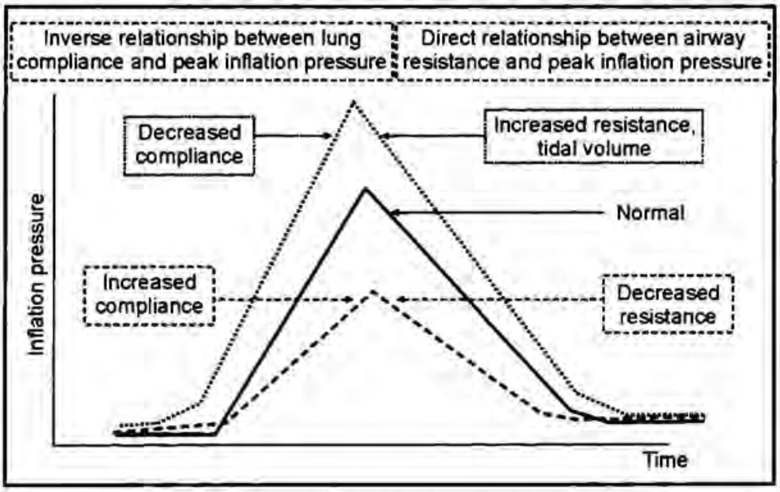


Figure 29.1: Factors influencing airway pressure in volume controlled ventilation

Alarm Limit

To protect the patient's lung from excessive pressure, it is very important to set the upper pressure limit to a suitable value. Pressure cycling serves primarily as a backup cycling criteria during flow controlled, volume or time cycled ventilation (when airway pressure reaches a preset alarm threshold). Plateau pressure should not exceed 30 cmH_2O.

Characteristics of the Volume Controlled Ventilation (Fig. 29.2)

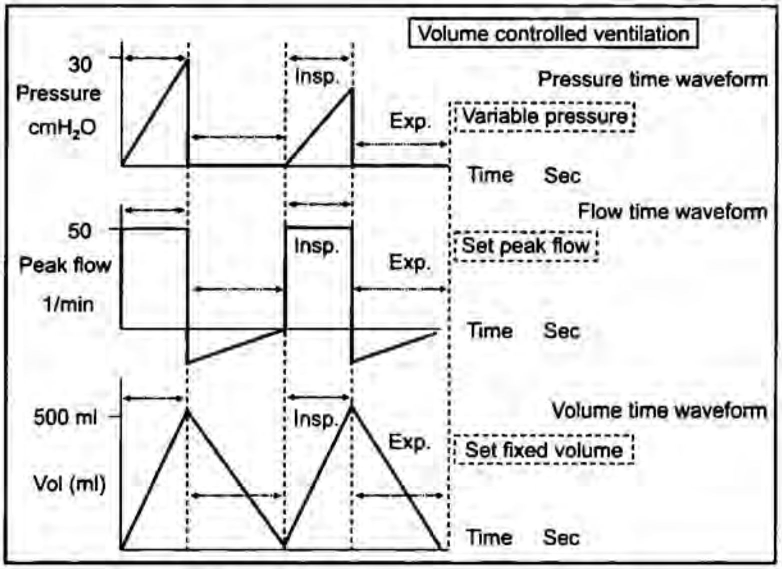


Figure 29.2: Waveforms in volume controlled ventilation

1. Volume controlled ventilation ensures that the patient receives a certain preset tidal/minute volume, during a preset inspiratory time
2. Inspiration starts according to the preset frequency or patient trigger
3. The inspiratory flow is constant
4. Expiration starts
   - When the preset tidal volume is delivered and after the preset pause time.
   - If the upper pressure limit is exceeded.

High Inspiratory Flow (Fig. 29.3)

The inspiratory flow is a measure for the velocity with which the breathing gas is delivered.

If ventilation occurs with a high inspiratory flow, then preselected tidal volume is delivered before the inspiratory time is completed, and

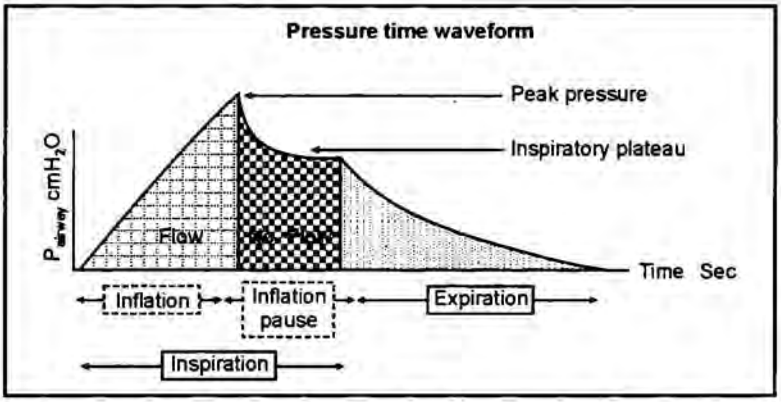


Figure 29.3: Pressure time waveform in volume controlled ventilation with high inspiratory flow

inspiratory time can be divided into a flow and no flow phase. In the pressure-time waveform of volume controlled ventilation with high inspiratory flow, an inspiratory pressure plateaus (no flow phase) will be seen (Fig. 29.4).

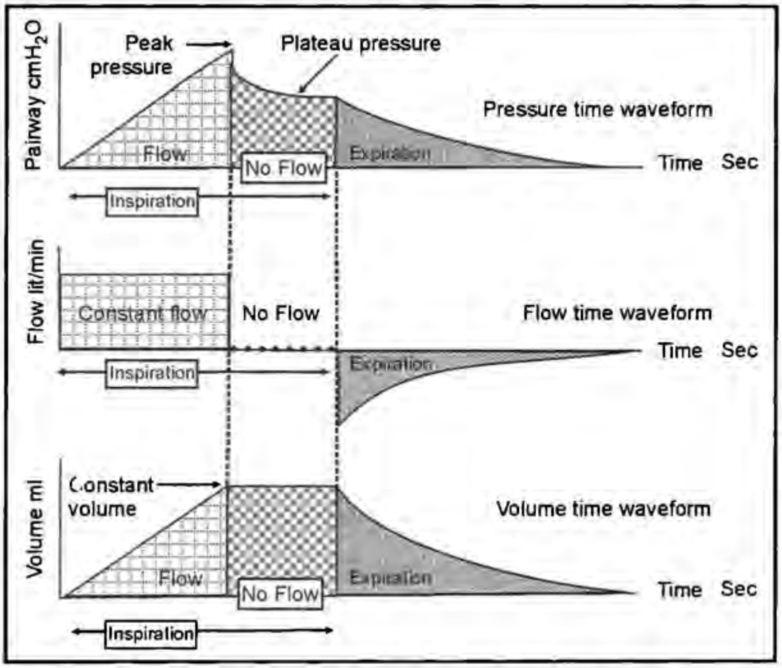


Figure 29.4: Various waveforms in volume controlled ventilation with high inspiratory flow

The increased in flow will increase ventilation pressure and also increases difference between peak and plateau pressure. Pressure peaks in the pressure time waveform disappears when the pre-selected inspiratory pressure approaches or falls below the plateau pressure (Fig. 29.5).

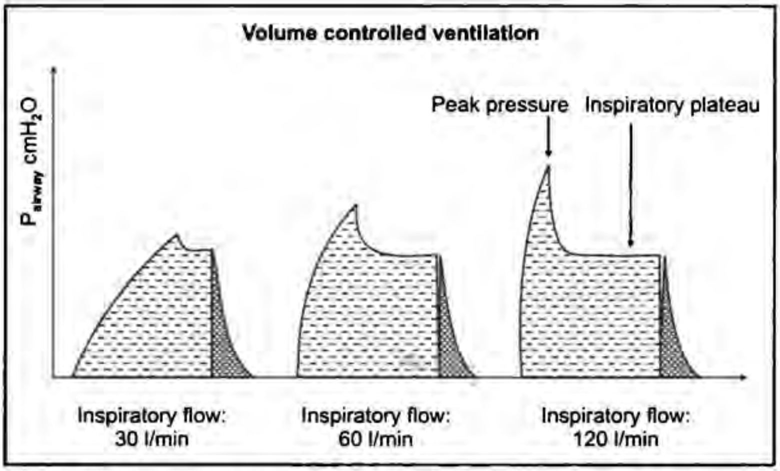


Figure 29.5: Ventilatory pressure changes with various inspiratory flow in VCV

The output variable in volume controlled ventilation is pressure, which depends on lung mechanics. The pressure waveform will change depending on pressure, which in turn depends on improvement or worsening of lung mechanics (Fig. 29.6).

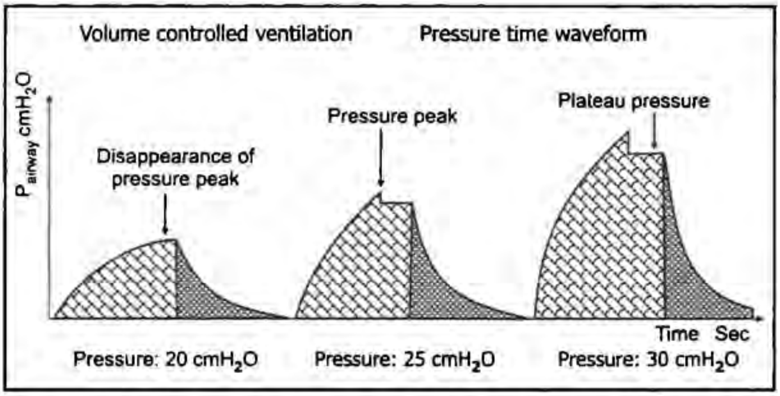


Figure 29.6: Change in pressure waveform with pressure changes in VCV

High Inspiratory Flow During VCV has the Following Effects

Barotrauma

High inspiratory flow will increase inspiratory peak airway pressure producing pressure induced lung injury.

Volutrauma

High inspiratory flow gives rise to overinflation of healthy lung compartments (short time constant) and compartments with increased compliance. However, in deranged respiratory mechanics and compartments with long time constant will have insufficient ventilation. This inhomogeneous ventilation leads to impaired ventilation/perfusion ratio and increased intrapulmonary right to left shunt.

Pendelluft (Fig. 29.7)

A group of identical breathing mechanical subunits of the lung is referred as compartment. For practical purpose model of lung can be divided into two compartments:

- *Short time constant*—A system with a short time constant will react very fast to a stimulus, it is called quick compartment
- *Long time constant*—A system with a long time constant will react very slowly to a stimulus, it is called slow compartment

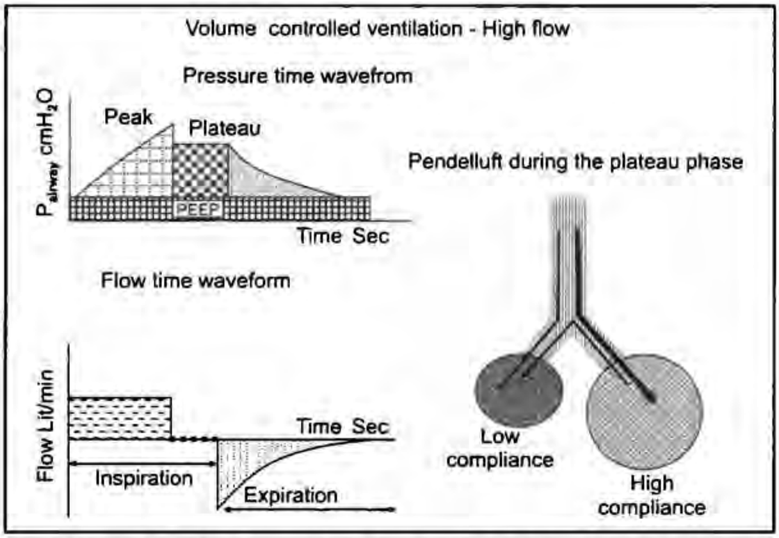


Figure 29.7: High inspiratory flow in volume controlled ventilation (For colour version *see* Plate 13)

Pendelluft represents an intrapulmonary redistribution of the breathing gas, i.e. the gas which has already taken part in the gas exchange, now flows from the quicker compartment (lung compartments with high compliance - a short time constant) to the slower compartment (lung compartments with low compliance - a long time constant). This breathing gas which is received by slower compartment, therefore having low in oxygen content. Pendelluft arises from pressure difference between the lung compartments in the inspiratory pause. In extreme case a compartment fills even when other compartment is still exhaling.

During volume constant ventilation with high flow, an endinspiratory plateau can be observed in pressure time waveform. Pendelluft arises during endinspiratory plateau of inspiratory phase, between compartments with considerably different time constants. The greatest part of the inspired volume is initially distributed to, the compartment with short time constant. During inspiration a pause or so called no-flow-pause, the lung remains inflated for a certain period, during which the administered gas volume is redistributed in the various compartment, depending on the different pressure gradients (due to different time constant).

During volume constant ventilation with low flow, an endinspiratory plateau is not observed in pressure time waveform and there is absence of no flow phase. Though there is different time constant with various compartments due to low flow pendelluft is not seen (Fig. 29.8).

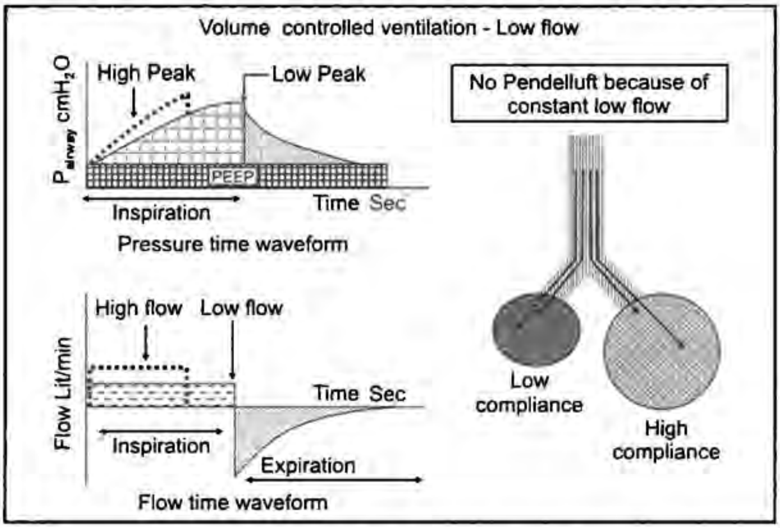


Figure 29.8: Low inspiratory flow in volume controlled ventilation (For colour version *see* Plate 13)

Volume Controlled Minimal Flow Ventilation

Ventilation with the lowest possible inspiratory flow is referred as "Volume controlled minimal flow ventilation. Even in volume controlled minimal flow ventilation a pressure limit must be set approximately 30 cm H_2O to avoid barotrauma (Fig. 29.9)

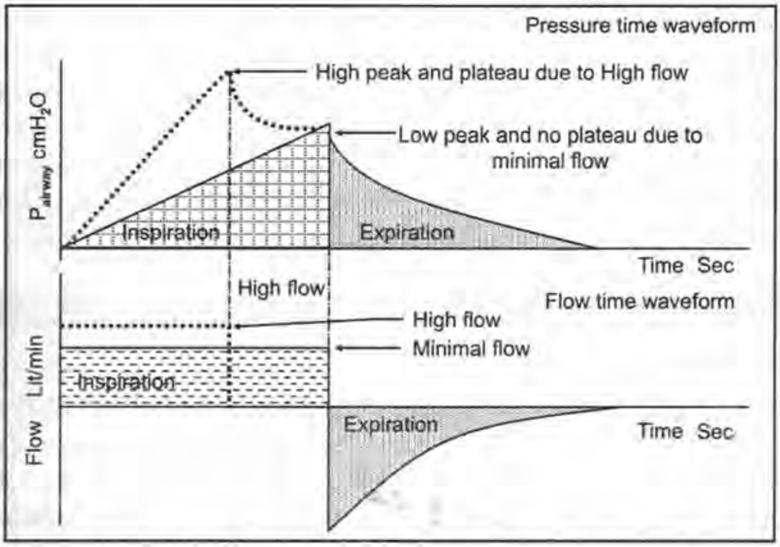


Figure 29.9: Minimal flow in volume controlled ventilation (For colour version *see* Plate 14)

Due to disadvantages of high inspiratory flow, the inspiratory flow level has to be set as low (minimal flow) as possible to:

- Keep ventilation as low as possible
- Keep the ventilatory pressure in the lung as low as possible
- Try to keep inspiratory pause (no flow phase) as short as possible

Disadvantages of Too Low Inspiratory Flow (Fig. 29.10)

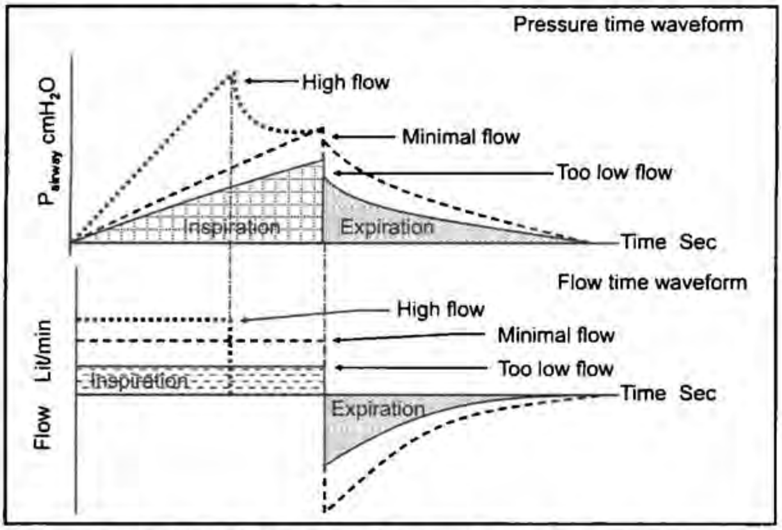


Figure 29.10: Too low flow in volume controlled ventilation (For colour version *see* Plate 14)

The selected volume cannot be administered in the selected time if the inspiratory flow is set too low. In VCV inspiratory time is constant and constant volume can not be delivered due to fixed inspiratory time. Thus volume is limited due to fixed inspiratory time, i.e. it no longer involves constant volumes. Low inspiratory flow rates would increase inspiratory time, but decrease expiratory time, which could lead to air trapping, auto-PEEP, patient discomfort and barotrauma.

INDICATIONS FOR VCV

Healthy Lung

Volume controlled (flow constant ventilation) is the preferred type of ventilation for the healthy lung. It is therefore administered mainly in anaesthesia. In anesthetic practice we administer volume controlled, pressure limited, time cycled ventilation that is usually limited to 35 cmH_2O in adults. The pressure limitation reduces the risk of barotrauma.

Craniocerebral Trauma

In craniocerebral trauma, flow constant ventilation guarantees safe administration of the selected tidal volume and therefore exact adjustment of the $PaCO_2$ is possible. The maintenance of normal range of $PaCO_2$, is vital to maintain normal intracranial tension.

Leaks in the Breathing System (Fig. 29.11)

Leaks in the breathing system or around the endotracheal tube lead to a steep decrease in tidal volume, particularly in volume constant ventilation.

By increasing the inspiratory flow, constant flow respirators can compensate for small to medium leaks. With larger leaks, the set inspiratory pressure is not reached, despite sufficiently high flow, and a reduced tidal volume is the result. Ventilation is sufficient, as long as an inspiratory plateau remains. However, during leak pressure controlled ventilation is better choice than volume controlled ventilation.

BENEFITS AND RISK

In contrast to pressure controlled ventilation, the flow rate is predictable and predetermined. By using the traditional flow controlled, volume/time cycled mode, minute ventilation is guaranteed safely over prolonged period of time, although pressure can rise significantly when respiratory

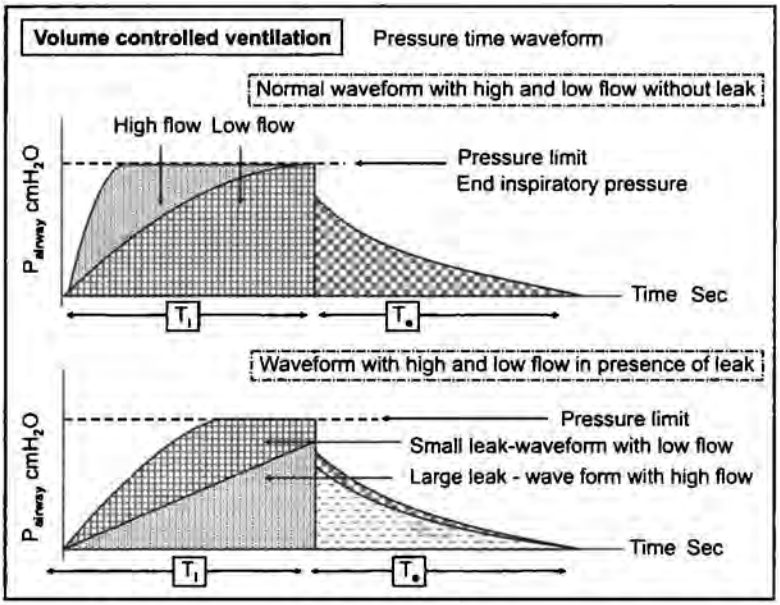

Volume controlled ventilation
Pressure time waveform
Normal waveform with high and low flow without leak
High flow
Low flow
Pressure limit
End inspiratory pressure
P_{airway} cmH$_2$O
Time Sec
T_I
T_e
Waveform with high and low flow in presence of leak
Pressure limit
Small leak-waveform with low flow
Large leak - wave form with high flow
P_{airway} cmH$_2$O
Time Sec
T_I
T_e

Figure 29.11: Compensation of minimal leak by increasing inspiratory flow

impedance increases. For a similar tidal volume and mean flow rate, the peak airway pressures may exceed, than those seen during pressure controlled ventilation depending on changes in lung mechanics, which is major disadvantage.

CHAPTER

30

Pressure Controlled Ventilation

INTRODUCTION

The ventilator regulates either the pressure applied in the airways (pressure controlled ventilation) or the pattern of flow delivery (volume controlled ventilation). The risk for ventilator induced lung injury due to large inflation volumes and risks of high inspiratory pressures (during volume controlled ventilation) has led to interest in pressure controlled ventilation. Because peak airway pressures can be kept lower in pressure controlled than in volume controlled ventilation, there is less risk of barotrauma with pressure controlled ventilation. The recent trend is 25% of patients receiving prolonged mechanical ventilation are managed on pressure controlled ventilation.

DEFINITION

Pressure-controlled ventilation (PCV) is completely controlled by the ventilator; it regulates the pressure applied in the airways with no participation by the patient.

COMPONENTS OF PRESSURE CONTROL VENTILATION (FIG. 30.1)

- *Type of breath – Mandatory/Control/Machine breath*
  Only mandatory breaths are available to the patients in the pressure control mode.
- *Triggering mechanism*
  The mandatory breaths in the pressure control mode are always triggered by a preset respiratory rate.

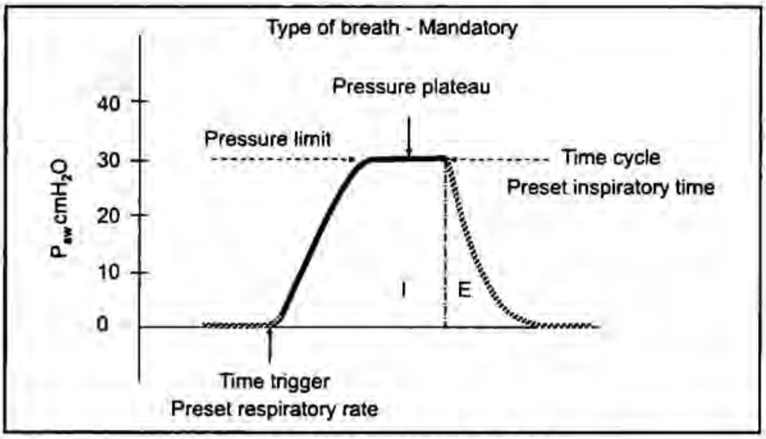


Figure 30.1: Components of PCV

- *Pressure limited*

  Once inspiration begins a pressure plateau is created and maintained for a preset inspiratory time.
- *Cycling mechanism*

  The mandatory breaths are time cycled by a preset inspiratory time.

Pressure controlled breaths are therefore time triggered, pressure limited and time cycled. Once the breath is initiated, these modes apply and maintain a targeted amount of pressure at the airway opening until a specified time (preset inspiratory time).

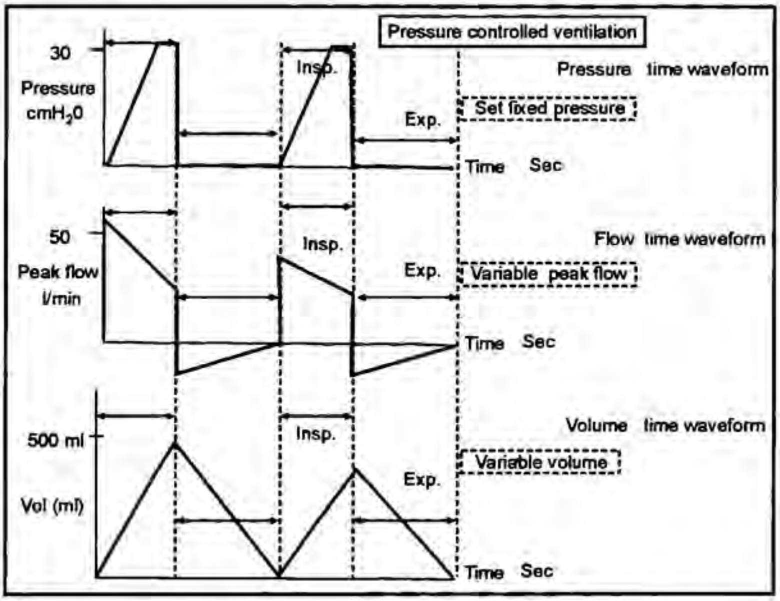


Figure 30.2: Waveforms in pressure control ventilation

WAVEFORMS IN PCV (FIG. 30.2)

Waveform can identify mode of ventilation. Set fixed pressure is seen in pressure time waveform of pressure controlled ventilation.

Variable peak flow is seen in flow time waveform, giving rise to variable tidal volume depending on respiratory mechanics. Tidal volume is the degree of freedom in pressure controlled ventilation.

The most important features of PCV is that maximal airway and alveolar pressures are restricted by the limit of preset pressure, whereas tidal volume, inspiratory flow, minute volume and alveolar ventilation depends on the impedance of the respiratory system.

APART FROM THE EXTERNAL PEEP, THE CLINICIAN SETS ONLY THE FOLLOWING PARAMETERS

Inspiratory Pressure – Pressure Level above PEEP

In pressure controlled ventilation, the breathing gas flows under preselected constant pressure (PIP or P_{max}) into the lungs during the selected inspiratory time and is maintained for the total duration of inspiration, though pressure exceeding 35 cm H_2O should generally be avoided. The flow is always highest at the beginning of inspiration (i.e. when the volume is lowest in the lungs). As the pressure is constant the flow is initially high and then decreases quickly with increasing filling of the lung (decelerating flow).

The necessary inspiratory pressure level to deliver tidal volume depends on the compliance of the lungs. In PCV ventilation the ventilatory pressure automatically adjusts and remains constant to the different mechanical characteristics of the lungs within certain pressure limits.

Inspiratory Rise Time or Inspiratory Pressure Slope (Fig. 30.3)

Machine varies in the rapidity with which airway pressure is built towards the preset maximum value. In some machines clinicians can adjust rise time to suit the situation whereas it adjusts automatically in some machines.

The inspiratory pressure level and the steepness of the pressure increase is set in PCV in a such way that on one hand the selected tidal volume is administered, on the other hand the initial inspiratory flow is not too high (< 2 lit/sec).

- *Faster rise time*
  Faster rates of pressurization are needed in certain situations when flow demands are high or during low impedance conditions (large patients) because of machine limitations.

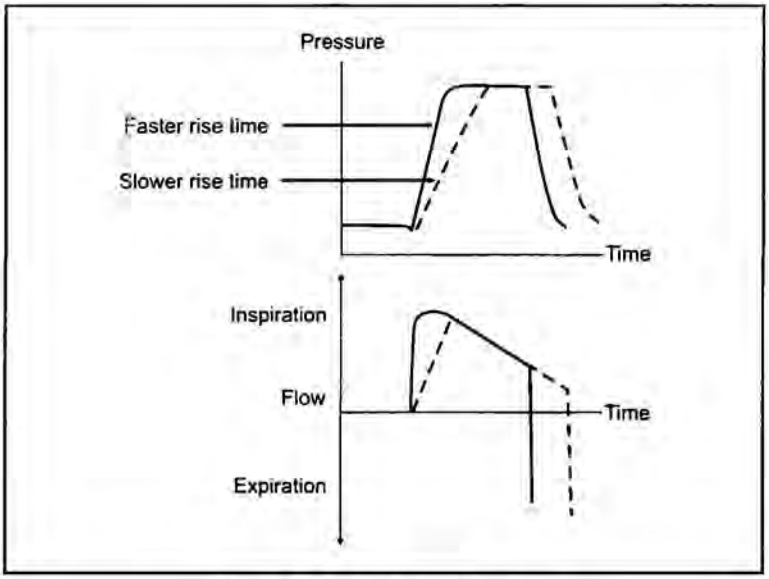


Figure 30.3: Inspiratory rise time

- *Gradual rise time*
  A more gradual pressure buildup is appropriate for quite breathing and during high impedance conditions.

Mandatory Frequency (respiratory rate)

The mandatory breaths in the pressure control mode are all the time triggered by a preset respiratory rate and time cycled by a preset inspiratory time. Over the lower frequency range, increasing frequency tends to improve alveolar as well as total ventilation, decreasing arterial $PaCO_2$. In a high frequency range, increase in frequency at fixed T_i/T_{tot}, curtails inspiratory time. Shortened inspiratory time prevents equilibration between applied airway and alveolar pressures which decreases tidal volume. As tidal volume decreases dead space ventilation increases, which will increase $PaCO_2$.

Breathing Cycle Time

- Inspiration starts according to the preset frequency or when the patient triggers.
- Expiration starts after the termination of preset inspiration time or if the upper pressure limit is exceeded. Pressure increases above the set upper pressure limit if the patient is coughing or the patient tries to exhale during the inspiration. Increase in pressure opens then expiratory valve and the ventilator switches to expiration.

Alarm Limits

As the delivered tidal volume can vary with change in respiratory mechanics it is very important to set alarm limits for tidal volume and minute ventilation to adequate levels.

OUTPUT VARIABLES OF PCV

1. **Inflation Volume**

   In pressure control ventilation the tidal volume is a degree of freedom, which will alter if there is a change in compliance or resistance for, e.g. if there is a sudden increase in bronchial resistance the patient hypoventilates. The peak inspiratory pressure (P_{max} or P_{peak}) is controlled by the preset pressure limit. The ventilator adjusts the inspiratory pressure (constant pressure) continuously to the ever changing compliance/resistance values. The volume delivery is dependent on applied pressure, i.e. pressure gradient existing between the airway opening and the alveolus at the onset of inflation.

Determinant of Tidal Volume during Pressure Controlled Ventilation

- *Effects of change in respiratory mechanics (Fig. 30.4):*
  The inflation volume increases as the peak inflation pressure increases (dashed lines). However, at a constant peak inflation pressure, the inflation volume decreases as the airway resistance increases (inverse

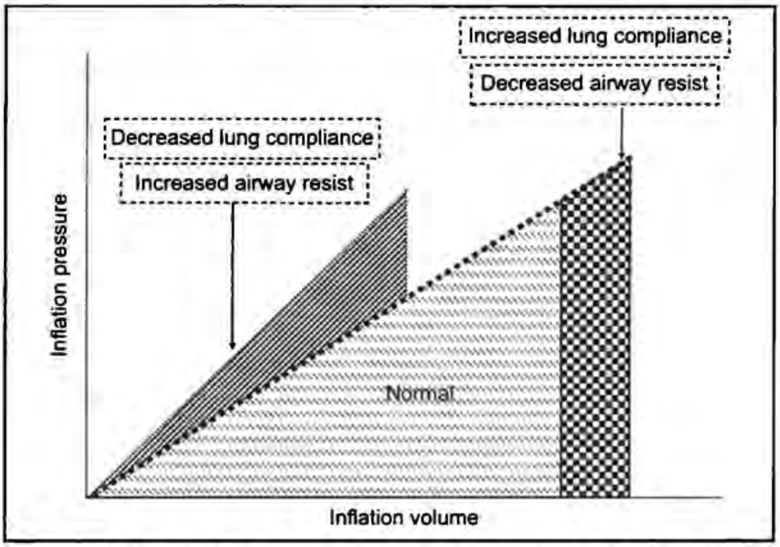


Figure 30.4: Effects of change in respiratory mechanics on tidal volume

relation) or the lung compliance decreases (inverse relation). In case of increased compliance less pressure is necessary to apply the same tidal volume.

Therefore, any change in lung mechanics during PCV can lead to a change in inflation volumes. As the delivered tidal volume can vary depending on respiratory mechanics, it is very important to set alarm limits for minute volume to adequate levels to safe guard safety of the patient.

- *Three times constant:*

  A three times constant is needed, for 95% of lung filling, thus maximizing delivered tidal volume, which also guarantees that alveolar pressures are in equilibrium with airway pressure, at the end of inspiration. In adult patient's equilibration requires 1-1.5 sec.

  Expiratory resistance is higher than inspiratory resistance; expiratory time should be longer than the three times constant, to empty the lung completely. Incomplete lung emptying is also an important factor affecting the delivered tidal volume.

- *Intrinsic PEEP:*

  Intrinsic PEEP presents a backpressure that opposes the applied pressure (P_{set}) and reduces the effective ventilating pressure. Effective ventilating pressure is equal to P_{set} - total $_{PEEP}$ (total $_{PEEP}$ = intrinsic $_{PEEP}$ + extrinsic $_{PEEP}$). Intrinsic PEEP increases with increasing frequency, reducing the effective ventilating pressure (P_{set} - total $_{PEEP}$) and consequently reducing tidal volume.

  The patient may also get auto-PEEP due to impedance, characteristic of the respiratory system. Higher frequency, longer Ti/Ttot ratio and higher P_{set} tend to increase intrinsic PEEP. The lower compliance and increased expiratory resistance, higher is the intrinsic PEEP for the same parameters. When the frequency increases at a constant value for P_{set} and T_i/T_{tot}, duration of inspiration and expiration both decrease and chances of intrinsic PEEP rises.

2. **Inspiratory Flow (Fig. 30.5)**

   In PCV pressure waveform is rectangular; with decelerating waveform for inspiratory flow, provided there is no patient effort.

   The inspiratory flow rate has a decelerating pattern determined as a function of driving pressure, airway resistance and respiratory system compliance. Hence, the flow rate cannot be adjusted independent of these variables; instead the inspiratory time is adjusted by a timing mechanism that cycles the ventilator between inspiration and expiration.

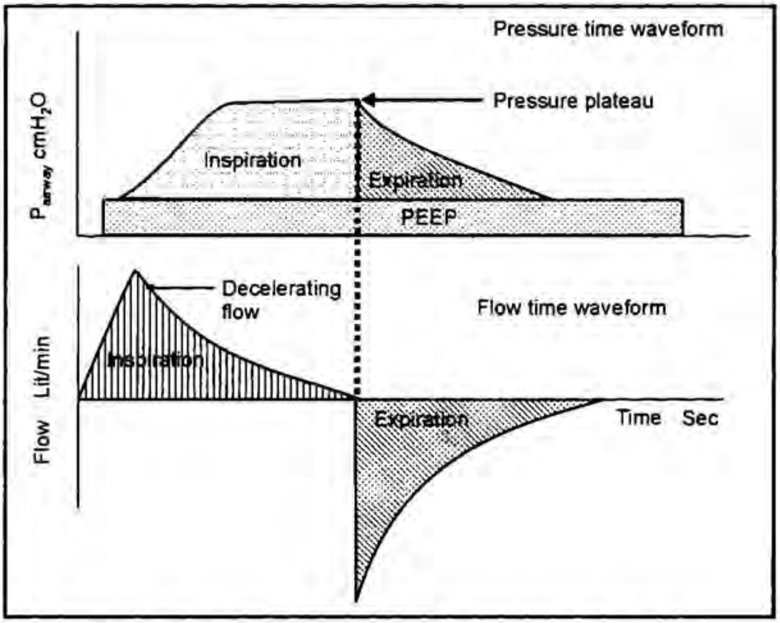


Figure 30.5: Decelerating waveform inspiratory flow in PCV

CHANGE FROM VCV TO PCV

The change from flow controlled, volume cycled ventilation (VCV) to PCV while keeping the same inspiratory time, resulted in more efficient elimination of CO_2 per breath, reflecting a lower dead space.

ADVANTAGES OF PCV

The major advantage of PCV relates to the following:

Decelerating Inspiratory Flow Pattern

In volume controlled ventilation, the inspiratory flow rate is constant throughout lung inflation. In pressure controlled ventilation, the inspiratory flow decreases exponentially during lung inflation (to keep the airway pressure at the preselected value).

- The decelerating inspiratory flow pattern reduces peak airway pressures and can improve gas exchange.
- Decelerating flow pattern allows the set pressure to be reached early during the breath and also allows pressure to remain constant throughout the inspiratory time and helps to improve the distribution of ventilation.
- It also helps to limit the end-inspiratory gradient of regional pressure among units with heterogenous time constants. When inspiratory

time is long enough, inspiratory flow may decrease down to zero before exhalation, a phenomenon that further favours distribution of air/pressure among heterogenous units and collateral ventilation.

Control of Airway Pressures

Under conditions of passive inflation, mean airway pressure reflects the average distending pressure of the respiratory system. Therefore mean airway pressure has been associated with two beneficial physiologic effects (ventilation and oxygenation) and potentially harmful side effects (hemodynamic compromise, barotrauma, etc).

Since in PCV, peak alveolar pressure cannot rise any higher than P_{set}. PCV is very convenient in critical patients when it is necessary to minimize ventilator induced lung injury. Transalveolar pressure rather than alveolar pressure is the key determinant of ventilator induced lung injury and barotrauma. In the absence of the patient's effort, keeping alveolar pressures within safe range is vital. Under these circumstances controlling airway pressure effectively controls maximal alveolar pressure.

INDICATIONS

Severe ARDS

Pressure control is usually indicated for patients with severe ARDS. During mechanical ventilation, patients with severe ARDS require extremely high peak inspiratory pressure in a volume controlled mode to maintain adequate oxygenation (PaO_2) and ventilation ($PaCO_2$). As result of these high airway pressures, they have a higher incidence of barotrauma in a normal lung. The advantage of switching these patients from volume controlled ventilation to pressure controlled ventilation is that the peak inspiratory pressure can be reduced while still maintaining adequate oxygenation (PaO_2) and ventilation ($PaCO_2$). Being able to decrease PIP significantly reduces the risk of barotrauma for these patients.

Leakage in the Breathing System

PCV is preferred when there is leakage in the breathing system. Capacity to increase the flow to maintain the preselected pressure can automatically compensate these losses well for small leaks. Therefore, it can be used with leaking cuff or uncuffed ETT tube, as in neonatal or paediatric patients. It is also useful in adult patients, in whom the airway cannot be completely sealed such as in bronchopleural fistula.

Spontaneously Breathing Patient with High Flow Demand

PCV can be used for spontaneously breathing patient with high flow demand that usually needs a high peak in the early part of the ventilatory cycle. This is only possible due to unlimited ability to deliver a flow and decelerating flow pattern.

Lung with Heterogenous Lung Properties

PCV is effective ventilation in cases of distribution disorders. The decelerating inspiratory flow associated with pressure limitation reduces overinflation of well ventilated faster alveoli (compartments with low resistance). Decelerating flow pattern improves the distribution of ventilation in a lung with heterogenous lung properties. The most rapid inspiratory flow occurring in the first part of inspiration with decelerating flow is most useful in dyspnoeic patients because flow demands are greatest during the earlier part of inspiration.

Normal Lung Mechanics—Neuromuscular Disease

PCV limits the lung exposure to high airway pressure producing barotrauma. Because of the influence of lung mechanics on inflation volumes during PCV, this method of mechanical ventilation seems best suited for patients with neuromuscular disease and normal lung mechanics.

To Control Ventilation and Oxygenation

- *Controlling pH and* $PaCO_2$
  This is done by controlling minute ventilation.
  - In volume control ventilation, setting of both the respiratory rate and the tidal volume can control pH and $PaCO_2$.
  - In pressure controlled ventilation, respiratory rate can be set, but tidal volume is indirect management depending on respiratory mechanics.
- *Controlling* PaO_2
  - This is done by adjusting the FiO_2, PEEP.
  - In volume control ventilation, peak inspiratory pressure is indirect variable depending on respiratory mechanics.
  - In pressure controlled ventilation, oxygenation can be improved by adjusting peak inspiratory pressure.

DISADVANTAGE OF PRESSURE CONTROLLED VENTILATION

There is an obvious disadvantage in not being able to guarantee V_T in a pressure controlled ventilated patient. During pressure controlled ventilation, V_T can rise or fall depending on compliance or airway resistance changes. The changes in mechanical properties are a common occurrence in unstable patients with an underlying respiratory disease. Due to the above reason in pressure support or pressure limited ventilation the V_T is no longer a preset parameter and it is mandatory to monitor V_T since it may reflect the progress or decline of lung mechanics.

Comparisons of Pressure Control and Volume Control Breaths (Figs 30.6 and 30.7)

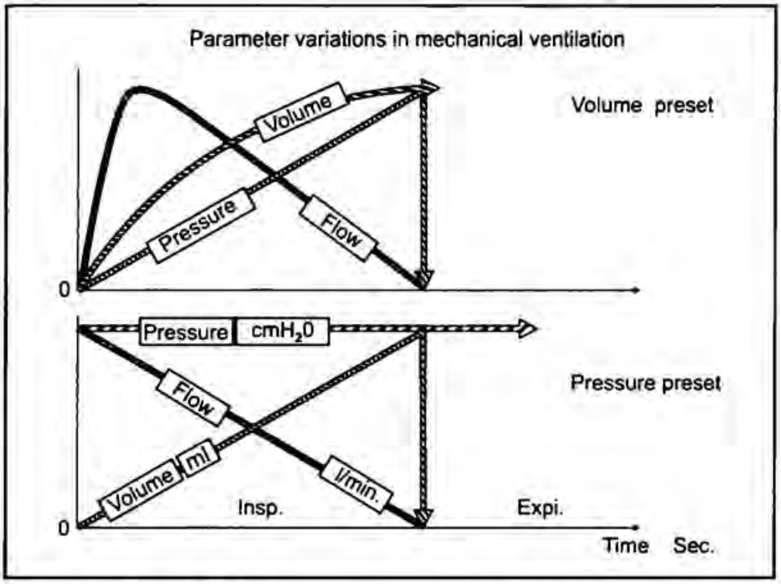


Figure 30.6: Parameter variations in PCV and VCV

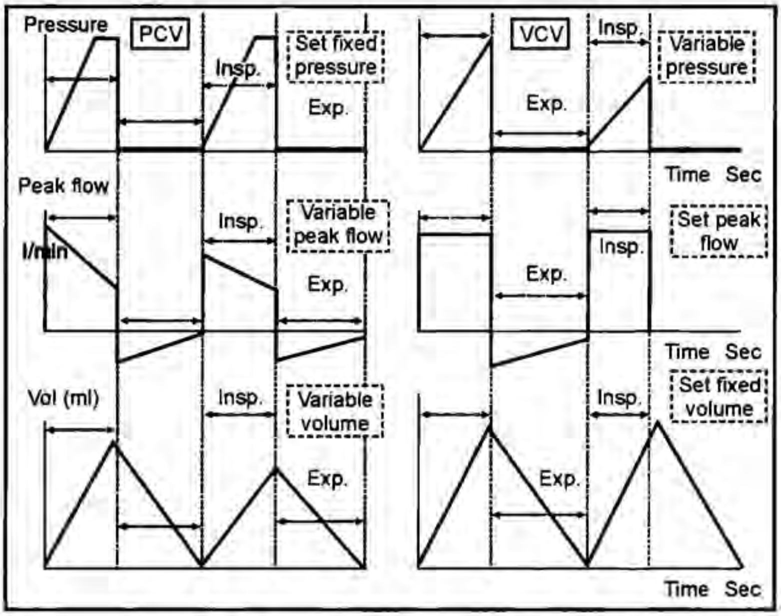


Figure 30.7: Waveform variations in PCV and VCV

Volume control breath	Variables	Pressure control breath
Set by clinician; remains constants	**Tidal volume**	Variable changes with respiratory system mechanics
Variable changes with respiratory system mechanics	**Peak inspiratory pressure**	Set by clinician; remains constant
Set directly or as a function of respiratory	**Inspiratory time**	Set by clinician; remains constant
frequency and inspiratory flow settings	**Inspiratory flow**	Variable with changes in patient effort and respiratory system impedance
Mostly constant flow wave form	**Inspiratory flow waveform**	Flow waveform is always decelerating.
Pressure waveform is always decelerating	**Pressure waveform**	Mostly constant pressure waveform

PRESSURE CONTROLLED VENTILATION AND PRESSURE SUPPORT VENTILATION

Pressure control ventilation has some functional similarities to pressure support ventilation, but they have different indications. Pressure-controlled breaths are time triggered by a preset respiratory rate, and once time triggered a pressure plateau is created and maintained by servo-controlled inspiratory flow for a preset inspiratory time. Pressure plateau in pressure support is maintained for as long as the patient maintains a spontaneous inspiratory flow.

PCV patient should be sedated since it is a control mode; however pressure support is used in spontaneously breathing patients no sedation is required. During prolonged use of pressure controlled airway pressure limits are guaranteed, although minute ventilation is at risk whenever there is change in lung impedance. In an attempt to improve safety or to decrease the need for repeated adjustments at the bed side new dual modes such as volume assured pressure support, pressure regulated volume control which have desirable characteristic of each category can be used.

CHAPTER

31

Synchronized Intermittent Mandatory Ventilation

SIMV is a mixture of mechanical ventilation and spontaneous breathing (Fig. 31.1).

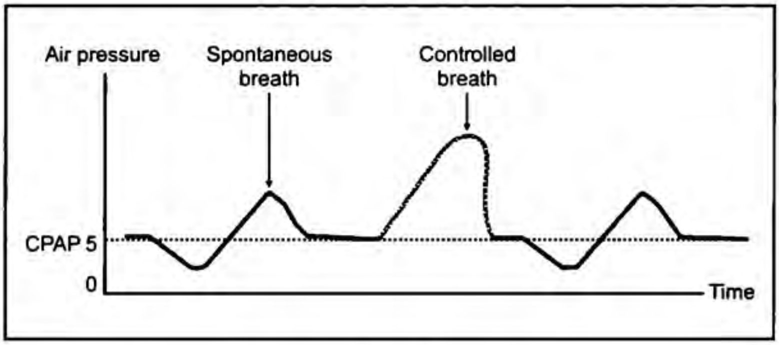


Figure 31.1: SIMV

It is a type of ventilator support in which a preset number of mandatory breaths are delivered while patient breathes spontaneously between mandatory breath (Fig. 31.2).

Apart from the number of mandatory breaths the ventilator pattern of the mandatory breaths can also be varied via the adjustable variables e.g. tidal volume, IPPV frequency, inspiratory flow. I:E ratio determines the duration of the mandatory breath, i.e. breath cycle time. SIMV mode uses a conditional variable to determine which type of breath to deliver, i.e. whether time or patient triggered. A mandatory breath ensures certain minimum ventilation to the patient. This minimum minute volume is determined by setting the tidal volume and f_{IMV}.

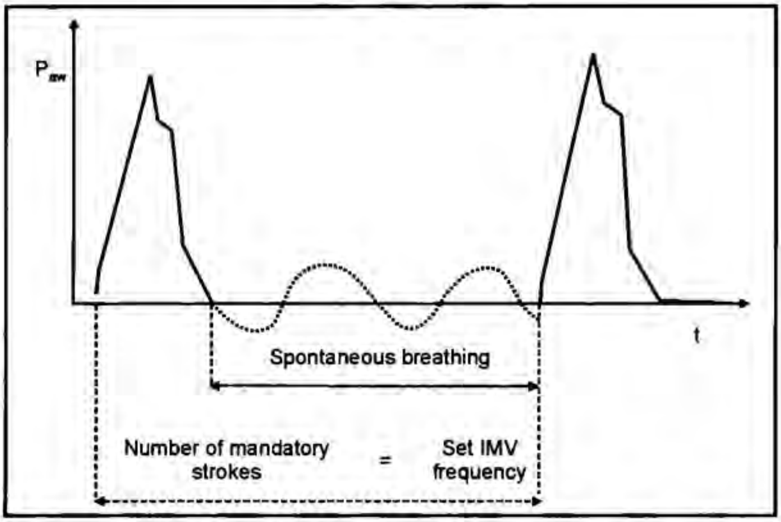


Figure 31.2: SIMV – Set IMV frequency

TYPES OF BREATHS IN SIMV MODE (FIG. 31.3)

- Time triggered mandatory breath is delivered automatically as a safety backup measure when the patient has no spontaneous effort.
- Assisted breaths are delivered to the patient at the beginning of inspiratory efforts thus preventing an increase in SIMV frequency.

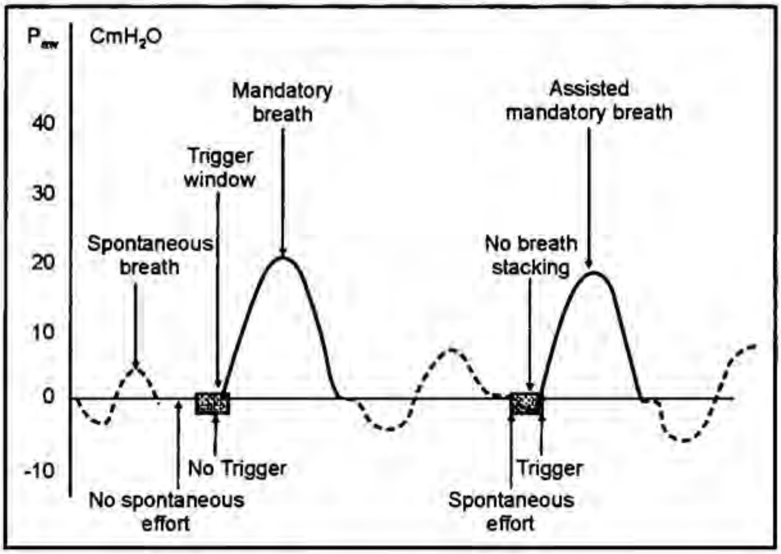


Figure 31.3: Types of breath

TYPES OF SIMV MODES

Depending on the parameter the SIMV breaths can be volume or pressure or dual controlled. Spontaneous breathing can be with pressure support (SIMV + Pressure support) with or without PEEP.

Mandatory Breaths

1. *SIMV (volume control) + pressure support* (Fig. 31.4)
    - Preset volume
    - Flow limited
    - Volume cycled or time cycled

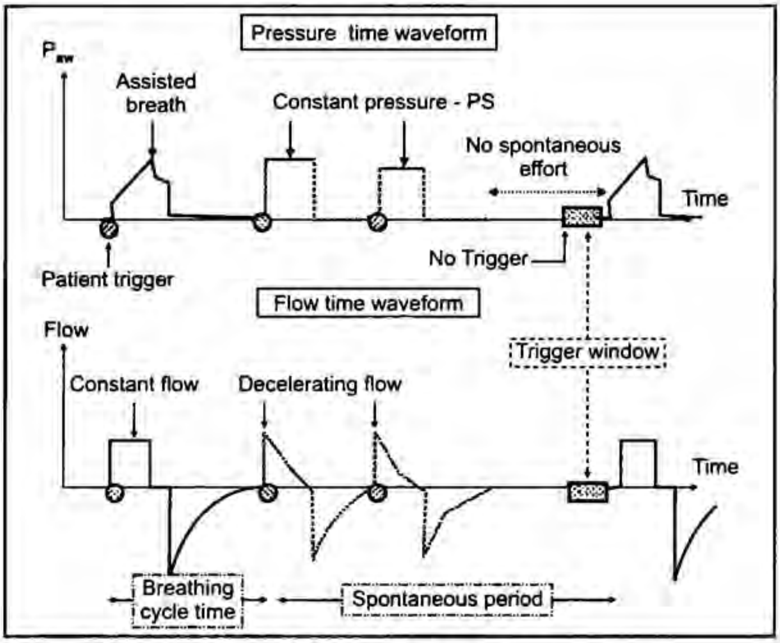


Figure 31.4: SIMV (Volume control) + Pressure support

2. *SIMV (pressure control) + pressure support* (Fig. 31.5)
    - Preset pressure
    - Pressure limited
    - Time cycled

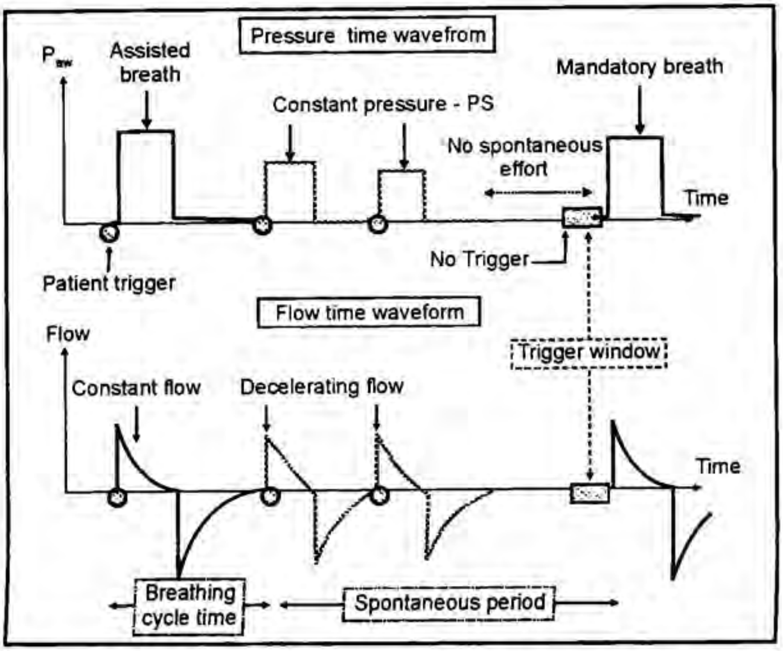


Figure 31.5: SIMV (Pressure control) + Pressure support

Spontaneous breaths

- Patient triggered
- Pressure support
- Patient cycled

SYNCHRONIZATION OR TIMING OR TRIGGER WINDOW (FIG. 31.6)

During SIMV the patient can breathe spontaneously and intermittently receive a number of mandatory ventilation strokes which correspond exactly to the set IMV frequency.

The time interval just prior to the scheduled delivery of the time-triggered mandatory breath is commonly referred to as the "Timing window or Synchronization window or Trigger window". Although the exact time interval of the synchronization window is slightly different from manufacturer to manufacture, 0.5 second is common time interval.

Mechanism of Synchronization (Fig. 31.7)

If the mandatory rate are selected is 10 breaths per minute, the ventilator would be expected to time trigger and deliver a mandatory breath every 6 seconds (set SIMV time).

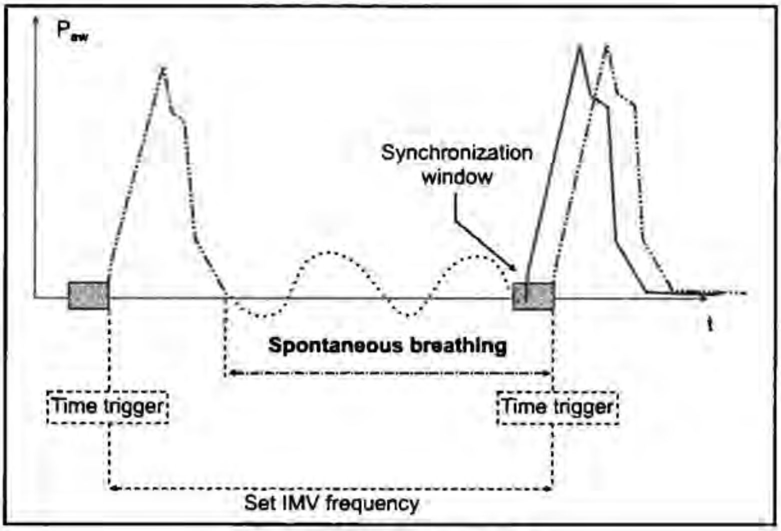


Figure 31.6: Trigger window

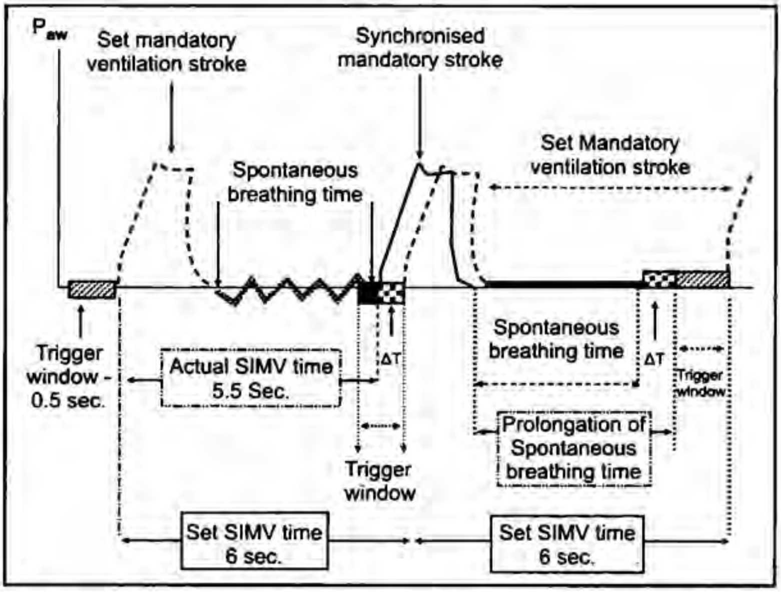

P_{aw}
Set mandatory ventilation stroke
Synchronised mandatory stroke
Set Mandatory ventilation stroke
Spontaneous breathing time
Trigger window - 0.5 sec.
Actual SIMV time 5.5 Sec.
ΔT
Trigger window
Spontaneous breathing time
ΔT
Trigger window
Prolongation of Spontaneous breathing time
Set SIMV time 6 sec.
Set SIMV time 6 sec.

Figure 31.7: Mechanism of synchronization (For colour version *see* Plate 15)

At 5.5 seconds from the beginning of the previous mandatory breath, the synchronization window (0.5 sec.) determines absence or presence of a spontaneous inspiratory effort.

Finely adjusted trigger mechanism (variable flow trigger) ensures that, within a trigger window, mechanical breath is triggered when the patient initiates inspiratory effort, thus SIMV mode does not cause breath stacking.

If the spontaneous breathing time is less than 5 seconds (instead of 5.5 seconds), the ventilator as a safety measure prolongs the subsequent spontaneous breathing time by the missing time difference "T, thus preventing an increase SIMV frequency. Thus mandatory breaths may be delivered slightly sooner or later than the preset time interval, however always within the time window. At higher IMV frequencies it can cover the total spontaneous breathing cycle.

Patient Triggering—Pressure/Flow

The demand valve is always patient triggered, either by pressure or flow depending on the settings. If the patient makes spontaneous inspiratory effort when the synchronization window is active, the ventilator is patient triggered to deliver an assisted mandatory breath. If however no spontaneous inspiratory effort exists while the synchronization window is active, the ventilator will time trigger when the full time triggering interval elapses to give mandatory breaths.

Spontaneous Breath

In between the mandatory breaths, SIMV permits the patient to breathe spontaneously to any tidal volume the patient desires. It is important to understand that the spontaneous breaths by the patient in the SIMV mode are truly spontaneous. The ventilator provides the humidified gas at the selected FiO_2, pressure support, CPAP, but the spontaneous rate and spontaneous tidal volume are totally dependent on the patient's breathing effort.

Significance of Setting Minimum Ventilation

The parameter f_{IMV} remains constant and together with tidal volume minimum ventilation can be set as a safety backup. If the patient has inhaled a significantly larger volume at the beginning of the trigger window, the machine reduces the subsequent mandatory ventilation stroke by shortening the time for the inspiratory flow phase and inspiratory time. In this way the other factor responsible for minimum ventilation, such as tidal volume remains constant.

ADVANTAGES OF THE SIMV MODE

Promotes Spontaneous Breathing

The most important advantage of the SIMV mode is that it promotes spontaneous breathing and use of respiratory muscles. Continuous use of respiratory muscles prevents respiratory muscle dysfunction. It is observed that prolonged skeletal muscle disuse leads to atrophy and perhaps to discoordination of diaphragmatic and accessory muscle contraction. SIMV helps to maintain respiratory muscle activity and strength because of continuous 'exercise' occurring with whatever level of spontaneous breathing the patient is able to maintain. PSV with SIMV is great help to the patient for preventing fatigue.

Reduced Need for Sedation

SIMV is used when the patient's condition is improved and the weaning process has to be started. During weaning process there is a need of maximization of spontaneous breathing, muscle relaxant or sedative drugs cannot be used.

SIMV Lowers Mean Airway Pressure (Fig. 31.8)

Mechanical breath increases airway pressure and spontaneous breath decreases it, any technique which is a combination of both will decreases mean airway pressure, reducing the risk of barotrauma and cardiovascular depression

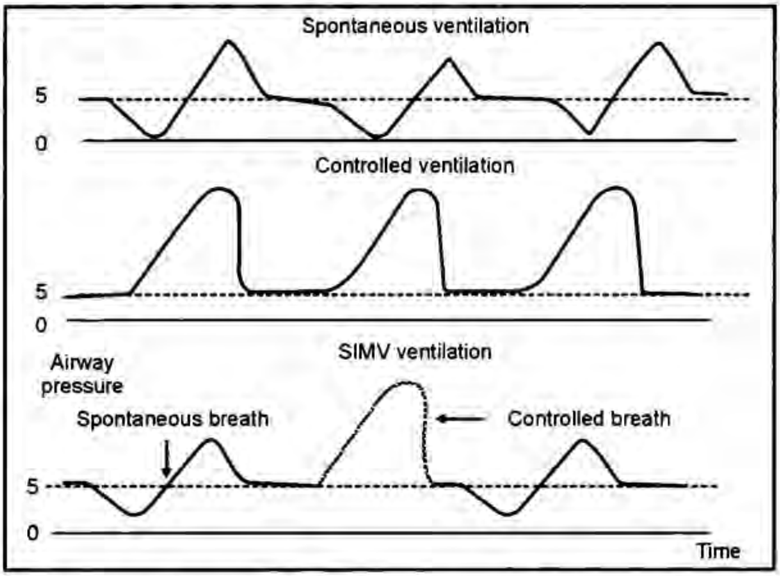


Figure 31.8: Comparison of pressure waveforms of various ventilation

Clinical Significance of Reduced Mean Airway Pressure

Indirectly enhances the patient's cardiovascular functions:
Reduction of the mean airway pressure during SIMV indirectly enhances the patient's cardiovascular functions. This minimizes the cardiovascular

effects of mechanical ventilation and less likely to cause a fall in the cardiac output and in arterial blood pressure.

Lesser chance of barotrauma / Volutrauma:
Fewer high peak inflation pressures and avoidance of large tidal volume of mechanical breath reduces the risk of pulmonary barotrauma significantly.

Improvement of Intrapulmonary Gas Distribution

In normal subjects a reduction of FRC averaging 500 ml occurs when they move from upright to supine. Patients who breathe spontaneously, ventilation and perfusion occurs in the dependent part, i.e. posteriorly. This will retain approximately normal V_A/Q matching inspite of reduced FRC.

If the diaphragm is rendered flaccid by drugs a V_A/Q relationship is altered dramatically. Most of the ventilation is directed to the nondependent regions while most of the perfusion remains dependent since pulmonary perfusion is gravity dependent. Hence, there is an increase of dead space anteriorly ($V_A>Q$), while an increase of shunting occurs posteriorly ($V_A<Q$).

Because of the decrease in mechanical breaths and increase in spontaneous breaths SIMV tends to reduce these ventilator-induced V_A/Q mismatch bringing them towards normal. Spontaneous breathing during SIMV tends to distribute the spontaneous tidal volume more evenly thus reducing alveolar dead space ventilation.

Avoidance of Respiratory Alkalosis

SIMV decreases severity of respiratory alkalosis compared to controlled ventilation. SIMV allows the mechanical breath to be delivered in response to the patient's spontaneous effort.

SIMV reduction in alkalosis results from the patient's ability to set both rate and tidal volume of spontaneous breathing, to meet his or her physiological requirements. The ventilator is used only to augment insufficient ventilation, bringing it to a normal level in terms of CO_2 elimination, hence normal pH and $PaCO_2$.

Facilitates Weaning

Of all the reported advantages of SIMV, weaning is perhaps the most controversial and hardest to establish. Weaning is not a major problem for most mechanically ventilated patients when the reason for ventilatory support no longer exists.

SIMV facilitates weaning with its ability to decrease the mandatory rate. This may offer some advantage to those "hard to wean" patients who cannot tolerate an abrupt decrease of the mechanical respiratory rate.

DISADVANTAGES OF SIMV MODE

Prolongation of Weaning

The ideal approach of SIMV weaning is to provide a spontaneous breathing workload that gradually increases a patient's muscle strength and endurance. SIMV with its gradual reduction of mechanical ventilation rate, allows some physicians to unnecessarily delay the weaning process.

Respiratory Muscle Fatigue

The primary disadvantage associated with SIMV is to desire to wean the patient too rapidly. Respiratory muscle conditioning may benefit from the careful use of SIMV but forcing the patient to breathe spontaneously when he or she is incapable of such effort leads first to an increased workload of spontaneous breathing, then ultimately to muscle fatigue and weaning failure. The best way to avoid this is to decrease the SIMV mandatory respiratory rate slowly and monitor the patient closely for signs of fatigue.

Increased Risk of Carbon Dioxide Retention

SIMV should be reserved for patients with stable central nervous system and respiratory control mechanism. Patients should not be unduly sedated or depressed with narcotics, muscle relaxants are absolutely contraindicated. Advantages of SIMV depend on the patient's capability for maintenance of spontaneous ventilation. At low ventilator rates, any decrease of this component can result in significant CO_2 retention and respiratory acidaemia.

INDICATIONS FOR THE SIMV MODE

To Provide Partial Ventilatory Support

There is an advantage to the patient in providing partial ventilator suppor, i.e. part of the minute volume.

Ease the Patient from Full Support to Partial Support

It is customary to ease the patient from full support to partial support by gradually decreasing the mandatory rate as tolerated by the patient. This depends on, the reversal of clinical conditions for which the patient was put to the ventilator.

Cardiovascular Instable Patient while on CMV with PEEP

Patient who develops hypotension and cardiovascular instability while on CMV with PEEP, SIMV with PEEP can be used, if clinical condition favours. The use of SIMV in such an individual is associated with a lower mean airway pressure, as this may restore cardiovascular stability.

Patient with Bronchopleural Fistula

A patient with respiratory failure associated with bronchopleural fistula, needs the lowest mean airway pressure that does not adversely affect ventilation and gas exchange, yet allows the bronchopleural fistula to heal and get sealed. The use of the SIMV mode may be ideally suited for such a patient, which can be combined with the lowest effective chest drainage tube suction.

SIMV can be Used for Long Term Ventilation

SIMV has reduced average ventilation pressure; it causes less stress on the circulation. The spontaneous breathing rhythm of the patient remains largely intact, so that there is less risk of ventilator dependency than with controlled ventilation. The basic idea of SIMV is that the patient breathes largely spontaneously and that the ventilator offers mechanical breaths with a very low safety frequency, so that minimum ventilation is ensured. Spontaneous breathing pattern remains, so lesser risk of ventilator dependency compared to controlled ventilation.

CONTRAINDICATION OF SIMV

- Critically ill patients with severe acute lung injury (ARDS)
- Haemodynamically unstable patients
- Patients with minute ventilation requirements > 10 l/min.

CHAPTER 32

Biphasic Positive Airway Pressure

The delivery of a preset tidal volume during IPPV has been seen as a very important feature for many years. This should guarantee unchanged ventilation even under conditions of changing lung mechanics. This concept has been questioned during the last decade. To avoid increasing airway pressures in response to a deterioration of lung mechanics, pressure-controlled ventilation has become more popular. PCV prevents an automatic rise of airway pressure if lung mechanics deteriorate and helps to avoid overdistension of the remaining, well-inflated lung areas.

CLASSIFICATION

Biphasic positive airway pressure (BiPAP) or Bi-vent is pressure-controlled ventilation which is a variant of APRV. In this mode the patient is allowed to breath spontaneously at two levels of CPAP. For this reason, BiPAP is often described as time cycled alteration between two CPAP levels. The only difference between BiPAP and Bi-vent is, pressure support for spontaneous breath can be used only to lower pressure level in BiPAP but both upper and lower level in Bi-Vent mode.

SPECIAL FEATURES (FIG. 32.1)

- In BiPAP the circuit switches between high and low levels of CPAP (airway pressure) in an adjustable time sequence. The two levels of CPAP are adjustable.

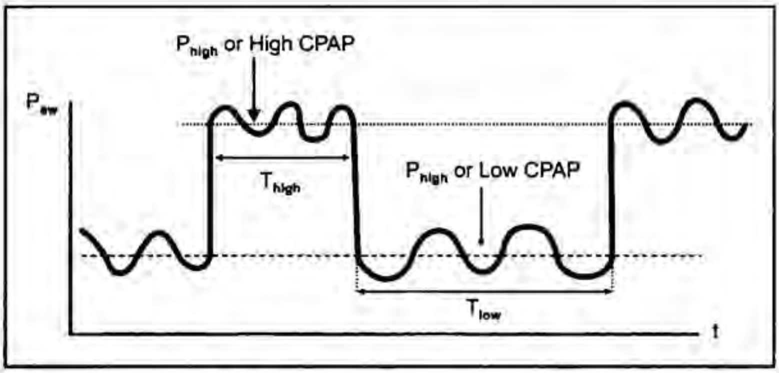


Figure 32.1: Biphasic positive airway pressure

At both pressure levels P_{high} and P_{low} the patient can breathe spontaneously. This is because the expiratory valve provides just enough gas required, for constant airway pressure via a regulatory mechanism, even for a small increase in the airway pressure.

The changeover from high CPAP to low CPAP synchronizes with the expiration of the patient while the pressure rise from low to high CPAP occurs during the inspiratory phase of the patient's spontaneous breaths, thus, the ventilator- patient asynchrony is prevented. (Fig. 32.2)

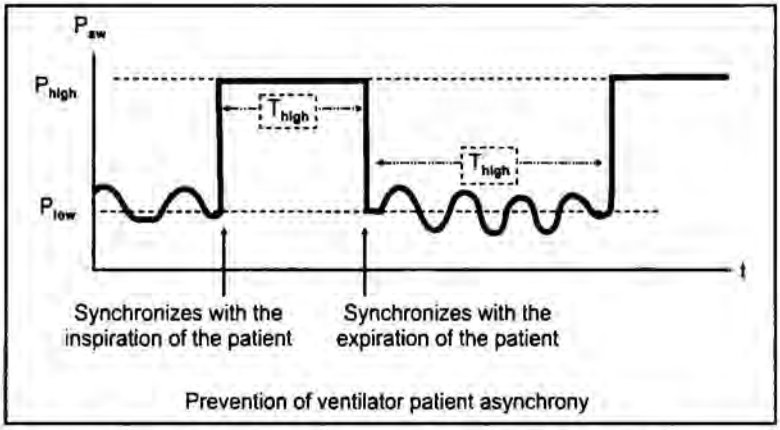

P_{aw}
P_{high}
T_{high}
T_{high}
P_{low}
t
Synchronizes with the inspiration of the patient
Synchronizes with the expiration of the patient
Prevention of ventilator patient asynchrony

Figure 32.2: Changeover from high CPAP to low CPAP

The mechanical part of ventilation results from the volume shift during the switching between the two airway pressures (CPAP) P_{high} and P_{low}. The resulting pressure difference causes gas flow. If the patient is not breathing spontaneously, this mechanical volume displacement is taken as pressure controlled mechanical ventilation.

VENTILATORY PARAMETERS WITH BIPAP VENTILATION (FIG. 32.3)

With BiPAP ventilation apart from inspiratory O_2 concentration there are only 4 adjustable variables

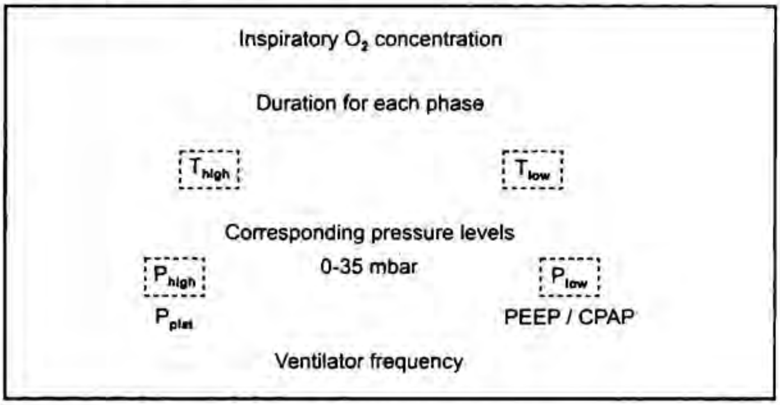


Figure 32.3: Ventilatory parameters

Duration for Each Phase

T_{high} (T_i) and T_{low} (T_e), i.e. the duration of higher and lower pressure, which is freely adjustable.

- *T_{high} or $T_{Inspiratory\ time}$ (T_i)*
  Inspiratory time results from the frequency and the $T_I : T_E$ ratio setting adapted to the patient's needs. When inspiration time T_{high} is long enough the pressure in the lung is P_{high}.
- *T_{low} or $T_{Expiratory\ time}$ (T_e)*
  T_{low} must be set long enough to allow the patient sufficient time to complete expiration.

  VF – Ventilator frequency, It is determined by T_i and T_e

$$VF = \frac{60}{(T_i + T_e)}$$

The I:E ratio and the ventilatory frequency can be adjusted to optimize ventilation and oxygenation.

Pressure Levels

The frequency at which the pressure changes are determined by IMV-frequency – f_{IMV}, so that the inspiratory time remains constant and only the length of the lower pressure level (CPAP) is changed.

Pressure Adjustibility

The pressure is adjustable from 0 to 35 cm H_2O

- *High CPAP or P_{high}*
  Only the P_{max} /P_{insp} setting needs to be changed to $P_{plateau}$ to switch over from the IPPV mode to the BiPAP mode as the time remains the same.
- *Low CPAP or P_{low} or PEEP*
- *Pressure support level above P_{low} or PEEP*
- *Pressure support level above P_{high}* (in Bi-vent mode only)

Steepness of Pressure – Inspiratory Rise Time

Steepness of increase from the lower pressure level to the upper pressure level can also be varied. The operation time for the increase in pressure cannot become greater than the set inspiratory time (T_i) thus guaranteeing that the upper pressure level is reached safely during inspiration.

Pressure Change

- *Lower to higher pressure level*
  This is activated with flow trigger (adjustable between 1-15l/min.) within a trigger time window. If there is no spontaneous breathing within this time window, at the end of the time window the ventilator switches to the upper pressure level.
- *Upper to lower pressure level*
  The change from the upper to lower pressure level happens when the patient begins to exhale, when the inspiratory flow has fallen to zero.

Alarm Limits for Minute Volume

The degree of freedom is tidal volume; consequently minute volume may not be constant. It is also very important to set lower and upper alarm limits for expired minute volume.

OUTPUT VARIABLES OF BiPAP

Tidal Volume

The tidal volume depends on:

- *Pressure difference*
  As in all pressure-controlled ventilation modes, the patient will not be having a fixed tidal volume. The tidal volume is principally from the pressure difference between the set values for P_{high} - $P_{low.}$ The display of the expiratory-measured tidal volume (V_{Te}) must be used

to set the required difference between the two pressure levels. An increase in the pressure difference results in a larger tidal volume.

- *Change in pulmonary mechanics*
  Change in lung compliance and airway resistance, as well as active "fighting" by the patient, can lead to changes in tidal volume.

Minute Volume

The delivered minute volume consists of the controlled mechanical ventilation caused by the sweep between the two pressure levels and spontaneous breathing.

MODES OF TOTAL AND PARTIAL VENTILATORY SUPPORT BY BiPAP

Depending on which of several possible different patterns of spontaneous breathing activity the patient adopts, the following ventilation patterns are covered by the BiPAP mode.

BiPAP - CMV (Fig. 32.4)

The time-cycled change of pressure gives controlled ventilation, which corresponds to pressure-controlled ventilation. The patient's spontaneous breathing activity is not possible at the upper CPAP level or at the lower CPAP level.

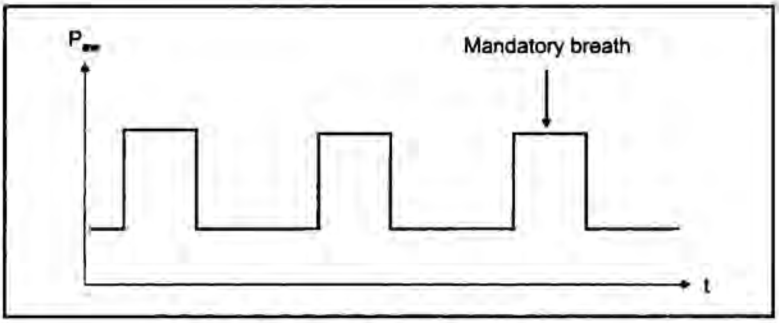


Figure 32.4: BiPAP CMV

BiPAP- SIMV (Fig. 32.5)

In accordance with the time adjusted, the patient receives his or her mandatory breath by switching to the upper pressure level. The patient is allowed to breath spontaneously to lower CPAP level.

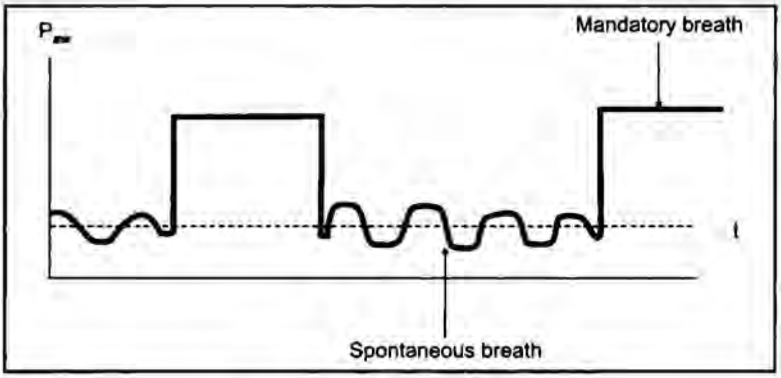


Figure 32.5: BiPAP – SIMV

BiPAP - SIMV offers the option of weaning through the frequency used SIMV concept and at the same time allowing the patient spontaneous breathing.

BiPAP – APRV Airway Pressure Release Ventilation (Fig. 32.6)

The ventilatory mode comprises CPAP that is intermittently released to allow a brief expiratory interval. The patient can breath spontaneously at the upper CPAP level under inversed ratio ventilation (IRV).

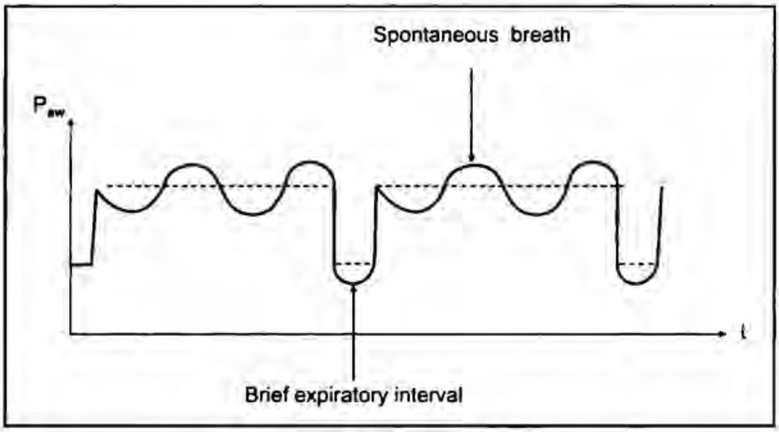


Figure 32.6: BiPAP – APRV

The brief reduction to the lower pressure level serves merely as ventilator support to improve CO_2 elimination. If you reverse the I:E ratio then you have APRV. Both modes can be applied with the same system. In contrast to APRV, which always implies an inversed ratio setting, BiPAP is not restricted to a special adjustment of the I:E ratio. It has been used in patients with ARDS and post-operatively in some patients and has been found to be effective in providing adequate oxygenation.

BIPAP - Genuine (Fig. 32.7)

In genuine BiPAP, spontaneous breathing is present, both at the upper and the lower CPAP level. This is superimposed on, but not impaired by, the mechanical component of ventilation.

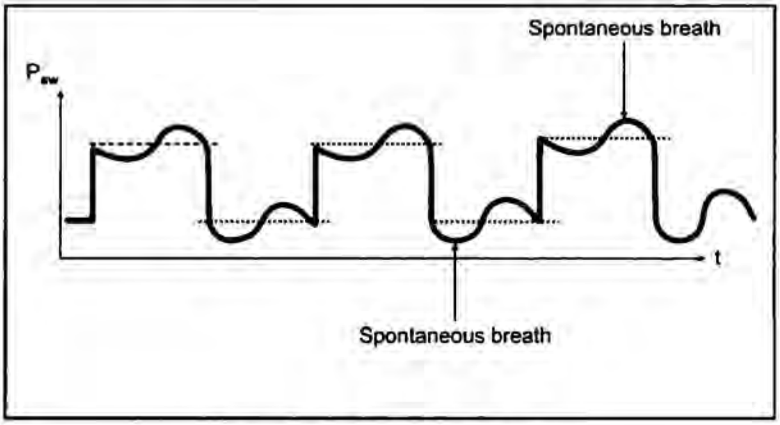


Figure 32.7: BiPAP – Genuine

CPAP (Fig. 32.8)

In this case "both" CPAP levels are identical, with continuous spontaneous breathing. Ventilation takes place exclusively by the spontaneous breathing activity of the patient.

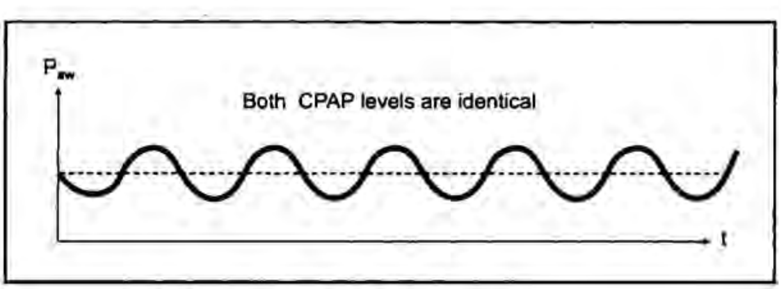


Figure 32.8: CPAP

BiPAP - Pressure Support (Fig. 32.9)

Pressure support gives additional support to spontaneous breathing at the lower pressure levels.

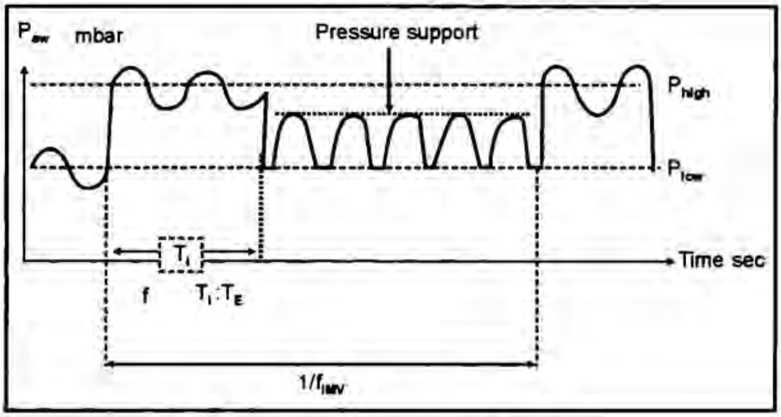


Figure 32.9: BiPAP - Pressure support

The other settings CPAP, P_{insp} and the steepness of pressure increase from lower to the upper pressure level are carried out in the same manner as for BiPAP. If pressure support is used to support spontaneous breathing at the lower pressure the same steepness of pressure increase comes into operation.

ADJUSTMENTS OF IPAP - P_{insp}

The upper pressure level controls peak airway pressure during inspiration. IPAP levels are generally determined by monitoring the patient's clinical and physiologic response to gradual changes of IPAP, rather by directly measuring the volume delivered. Increasing IPAP enhances the "pressure boost" and the rhythmic changes in FRC improves alveolar ventilation which helps:

- Improves hypoxaemia
- Normalize $PaCO_2$
- Reduces the work of breathing

Since BiPAP is not volume-cycled ventilation, a larger delivered volume may be obtained by:

- Increasing the IPAP level
  Decreasing the EPAP level
- Increased compliance
- Reduced flow resistance

ADJUSTMENTS OF EPAP

- Controls end-expiratory pressure
- Used as CPAP when IPAP = EPAP
- Used as PEEP when IPAP > EPAP

The EPAP can be increased by 2 cm H_2O increments to:

- Increase functional residual capacity and oxygenation in patients with intrapulmonary shunting
- Enhancing alveolar recruitment

PRECAUTIONS

Since IPAP and EPAP are methods to manipulate the airway pressure ventilation, all adverse effects of positive pressure ventilation and PEEP should be monitored. The weaning patient should be advised to report unusual chest discomfort, shortness of breath, or severe headache when using the BiPAP system.

DEPENDING ON THE CONDITION OF THE PATIENT, THE INITIAL PATTERN OF BIPAP VENTILATION COULD BE SET AS FOLLOWS

Initial Settings

Mode Selection

It depends on a patient's needs and ability to breath spontaneously since the BiPAP system in spontaneously breathing patient may be used in one of three modes.

1. Spontaneous – If the patient is breathing spontaneously
    - IPAP may be set at 8 cm H_2O
    - EPAP may be set at 4 cm H_2O
2. Spontaneous/timed mode is used as a backup mechanism

    The breaths per minute (mandatory breath) are set two to five breaths below the patient's spontaneous rate.

    Settings of timed mode (mandatory breath) – Backup
    - IPAP may be set at 8 cm H_2O
    - % IPAP may be set at
        1. 33% for an I:E ratio of 1:2
        2. 50% for an I:E ratio of 1:1
    - EPAP may be set at 4 cm H_2O
3. Time mode or Control mode
    - *Upper pressure level P_{high} (P_{insp}, IPAP)*

      12-15 mbar over PEEP depending on the estimated compliance of the patient. (About 20-22 cm H_2O)

The resulting tidal volume can be increased or decreased by raising or lowering P_{high} in the desired way.

- *Lower pressure level – P_{low} (PEEP, EPAP)*
  It is adjusted in accordance with the desired PEEP – 6-8 cm H_2O
- *The rate of breathing and the I:E ratio of BIPAP* ventilation are determined by:
  - The inspiratory time - T_{high} - 2 seconds
  - The expiratory time - T_{low} - 4 seconds

Setting Ventilator Parameters of the Patient who was on VCV (Fig. 32.10)

At the time of admission to the ICU, many artificially ventilated patients have prior history of volume-controlled positive pressure ventilation. Ventilator parameters are chosen depending on the present parameters of VCV.

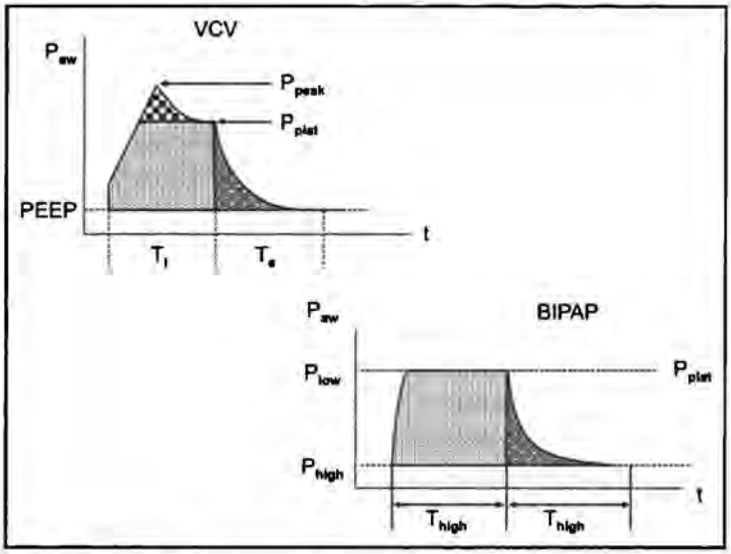


Figure 32.10: Setting ventilator parameters for BiPAP

1. *Upper pressure level – P_{high}*
   The plateau level of the VCV becomes the upper pressure level P_{high}.
2. *Lower pressure level - P_{low}*
   The lower pressure level corresponds to the PEEP value
3. *Time upper level T_{high} (T_i) and Time lower level (T_e)*
   T_{high} and T_{low} are adjusted in accordance with the inspiratory times and expiratory times of the volume-controlled mechanical ventilation (T_{high} – inspiratory time and T_{low} – expiratory time). In this way there is the same breathing rate and the same I:E ratio under BiPAP

as under the previous positive pressure mechanical ventilation. If an initial breathing frequency of 10/min and an I:E ratio of 1:2 time for upper pressure level is 2 seconds and that for the lower pressure level is 4 seconds.

4. *Tidal volume*

   The tidal volume under BiPAP is adapted to that under the volume controlled mechanical ventilation by variation of P_{high} by 1 cm H_2O above or below the same pressure level.

IMPORTANT FACTS ABOUT WEANING FROM ARTIFICIAL VENTILATION USING BiPAP

The "invasiveness" of mechanical ventilation, can be decreased according to the condition of the patient, the same way it is also possible to reduce the "invasiveness" linearly in the BiPAP mode.

1. *Unrestricted spontaneous breathing*

   As soon as the patient is ventilated with BiPAP, the patient has, in principle, the possibility of unrestricted spontaneous breathing irrespective of the phase of mechanical ventilation. Spontaneous breathing does not lead to fighting between the patient and the ventilator. Further relaxation is not required in any case.

2. *Reduce sedation*

   If there are no contraindications regarding spontaneous breathing in terms of the underlying disease, sedation can be reduced slowly in order to increase the contribution of spontaneous breathing to the ventilation, without having to change the mode of ventilation. This provides great comfort both for the patient and for the therapist during weaning. It is special help for the difficult decision as to when to change from controlled to an augmented ventilation mode.

3. *Tachypnoea – increase the depth of sedation*

   If the patient develops tachypnoea under reduced sedation, one only has to increase the depth of sedation. It is not necessary to switch to a "controlled" ventilation mode in this phase of mechanical ventilation

4. *Reduce FiO_2*

   The FiO_2 should first of all be reduced. A FiO_2 of less than 0.5 is desirable.

5. *Change parameter*

   During the weaning phase, the transition from controlled ventilation, to complete spontaneous breathing is achieved by a gradual reduction of inspiratory pressure, and/or frequency depending on an improved lung mechanics and gas exchange situation. If the patient is sufficiently awake, cooperative after an appropriate reduction of sedation, further weaning can take place.

6. *Change I:E ratio to 1 – under IRV*

   After reduction of the FiO_2, if the patient is on IRV, I:E ratio to 1 is aimed. In most cases the intrinsic PEEP phenomena is manifested under IRV ventilation, which will abolish once normal I:E ratio is maintained. Intrinsic PEEP can be observed without direct measurement of the intrinsic PEEP by examination of the expiration curve. If there is no end-expiratory "rest-flow" it can be assumed that no intrinsic PEEP phenomena are manifested.

7. *Reduce mean airway pressure*
   - PEEP should be reduced stepwise to a level of 7-9 cm H_2O.
   - By equidirectional reduction of P_{high} in accordance with the PEEP reduction, the mean airway pressure can be diminished in the same way.
   - The next step is a reduction of the pressure amplitude between P_{high} and P_{low}.
   - A pressure difference between 8 and 12 cm H_2O between the two CPAP levels should be aimed for depending on the compliance and resistance of the patient's lungs and airways.
   - The duration of the two pressure levels is are extended as the last step in weaning in the direction of CPAP.

8. *Lower frequency of mechanical ventilation*
   - Attain T_{high} and T_{low} between 3 and 3.5 seconds.

     After attaining a T_{high} and T_{low} between 3 and 3.5, T_{low} is subsequently lengthened exclusively. This results in a further lowering of the frequency of mechanical ventilation.
   - Mechanical ventilation of 4 bpm - T_{low} of 11.5 s with a simultaneous T_{high} of 3.5 s

     Depending on the situation in the individual patient, it is desirable to attain a frequency of mechanical ventilation of 4 bpm, which corresponds to a T_{low} of 11.5 s with a simultaneous T_{high} of 3.5. s.

9. *Switch to CPAP*

   After this weaning step the patient can be switched without any difficulty to spontaneous ventilation with CPAP. The CPAP level must correspond to the mean airway pressure, effectively present during the last BiPAP adjustment. CPAP prevents decrease in oxygenation due to reduced FRC, if associated with lowered mean airway pressure.

10. *Reduce CPAP level – 1-2 cm H_2O*

    Later, the CPAP level is reduced stepwise (1-2 cm H_2O) to a level between 5 and 7 cm H_2O, while paying attention to lung mechanics and gas exchange. If the patient is awake and the vital parameters are stable, extubation can be carried out.

ALTERATION OF BIPAP ADJUSTMENTS DEPENDING ON BLOOD GAS ANALYSIS

When alteration of BiPAP adjustments is required according to blood gas analysis, a distinction must always be made between—

- *Oxygenation failure*

  An increase in the mean airway pressure is to be aimed initially for failure of oxygenation, in order to achieve an increase of FRC and thus an increase in the area available for gas exchange.
- *Ventilatory failure*

  In the presence of ventilatory disorder (hypercapnia or hypocapnia) an increase or decrease of mechanical ventilation is necessary.

If the PaO_2 is low, oxygenation can be improved as follows: There are likewise two ways of increasing the mean airway pressure to counter hypoxia and optimize oxygenation by a corresponding gain of FRC, without altering the proportion of mechanical ventilation.

- *By equidirectional alteration of the upper and lower airway pressure level and change of airway pressure with unaffected durations* (Fig. 32.11)

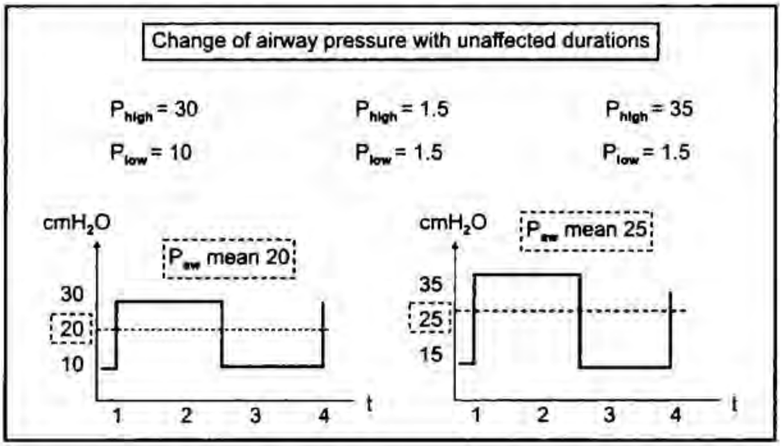


Figure 32.11: Equidirectional alteration of the upper and lower airway pressure level

Increase of the lower pressure level (P_{low}) and the upper pressure level (P_{high}) in the same value will an increase of the mean airway pressure.

This equidirectional alteration will not affect the contribution of mechanical ventilation, and will not change duration of ventilation. ΔP remains unchanged, therefore no change in the ventilation.

- *By Counterdirectional alteration of the duration of upper and lower airway pressure level and change of duration with unaffected airway pressure* (Fig. 32.12)

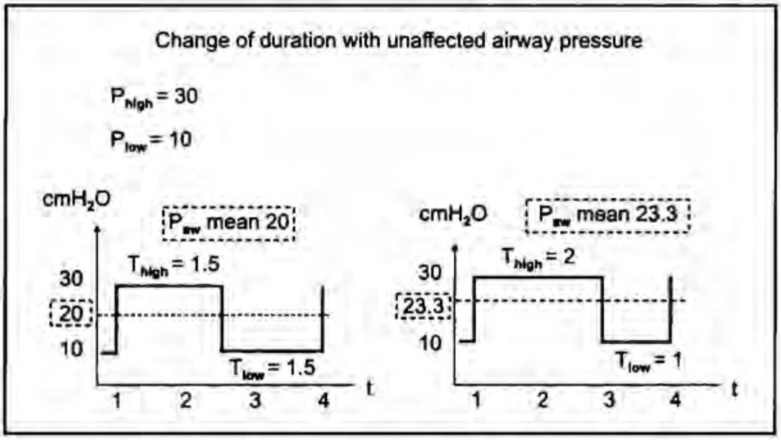


Figure 32.12: Counterdirectional alteration of the duration of upper and lower airway pressure level

By lengthening T_{high} and shortening T_{low} by the same amount and the consequent alteration of the pre-existing I:E ratio, there is an increase of the mean airway pressure which does not affect the contribution of mechanical ventilation. This is only valid if there is no intrinsic PEEP phenomenon after shortening T_{low}.

If the patient's gas exchange deteriorates subsequently, it is possible to increase the 'invasiveness' of ventilation at any time without having to change the mode of ventilation.

If the $PaCO_2$ is too high or too low, the ventilation is corrected by the following means

In the presence of hypercapnia with corresponding hypoventilation, mechanical ventilation is reduced in the opposite way:

- *Equidirectional alteration of the duration of upper and lower airway pressure level* (Adjustment of increasing ventilation with constant mean airway pressure) (Fig. 32.13)

  Shortening of T_{high} and T_{low} while maintaining the pre-existing I:E ratio brings about an increase of ventilation which has no effect on the mean airway pressure.

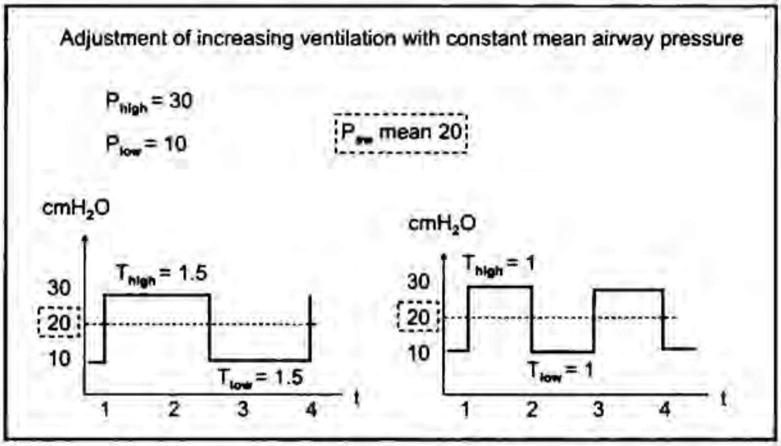


Figure 32.13: Equidirectional alteration of the duration of upper and lower airway pressure level

- *Counter-directional alteration of the upper and lower airway pressure level* (Adjustment of increasing ventilation with constant mean airway pressure) (Fig. 32.14)

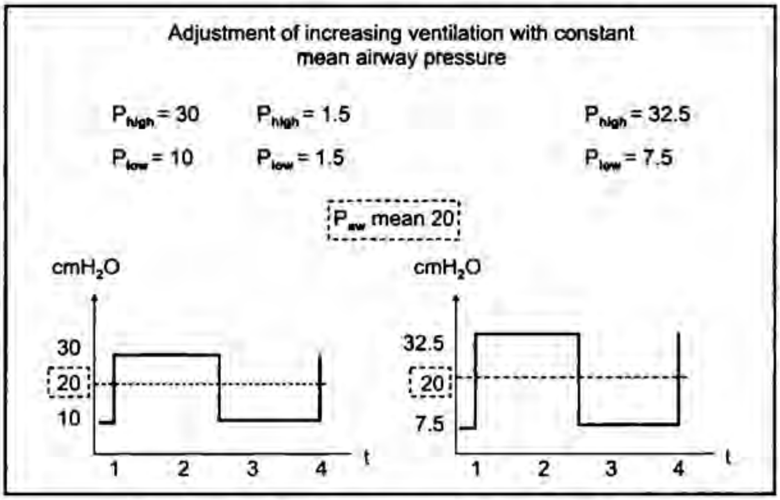


Figure 32.14: Counter-directional alteration of the upper and lower airway pressure level

By raising P_{high} and lowering P_{low} by the same value, an increase of mechanical ventilation can be achieved which does not affect the mean airway pressure.

This is valid for a given I:E ratio of 1:1. Under conditions of inversed ratio ventilation, e.g. I:E = 2:1, one has to lower P_{low} twice the value one has to raise P_{high} to get an unchanged mean airway pressure.

Summary to improve oxygenation and ventilation (Figure 32.15)

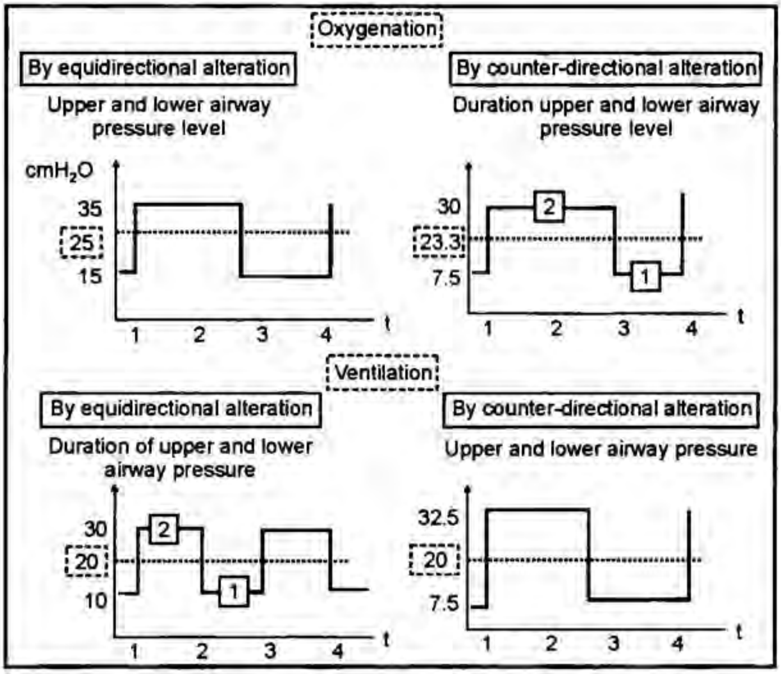

Oxygenation
By equidirectional alteration
Upper and lower airway pressure level
cmH₂O
35
25
15
1
2
3
4
t
By counter-directional alteration
Duration upper and lower airway pressure level
30
23.3
7.5
2
1
1
2
3
4
t
Ventilation
By equidirectional alteration
Duration of upper and lower airway pressure
30
20
10
2
1
1
2
3
4
t
By counter-directional alteration
Upper and lower airway pressure
32.5
20
7.5
1
2
3
4
t

Figure 32.15: Summary to improve oxygenation and ventilation

PaCO$_2$ Too High with Corresponding Hypoventilation

- *Increase the upper pressure levels*
  In the presence of hypercapnia with corresponding hypoventilation, mechanical ventilation should be increased.
  - *Increase in the pressure difference P_{high} and P_{low},* which will increase the tidal volume. The upper pressure level should lie below the upper inflection point of the pressure volume curve, to any avoid increase in the functional dead space.
  - *Shortened T_{high} and T_{low}* with maintenance of the pre-existing I:E ratio, which will increase the ventilator rate. Increase the breathing frequency synchronizing shortening of the phase times.

PaCO$_2$ Too Low with Corresponding Hyperventilation

- *Reduction of the upper pressure levels*
  In the presence of hypocapnia with corresponding hyperventilation, mechanical ventilation is reduced in the opposite way:
  - *Reduction in the pressure difference P_{high} and P_{low},* which will reduce the tidal volume

- *Prolongation of T_{high} and T_{low}* with maintenance of the pre-existing I:E ratio, which will reduce the ventilator rate.

ADVANTAGES OF BIPAP

1. *Option of spontaneous breathing*

   The great advantage of BiPAP in invasive ventilation regimes compared to continuous positive pressure ventilation (both pressure-controlled and volume-controlled) is the possibility of unrestricted spontaneous breathing irrespective of the phase of mechanical ventilation.

   In BiPAP weaning is a continuous process. The constant option of spontaneous breathing allows the transition from controlled ventilation (during weaning phase), to independent spontaneous breathing smoothly without any change in the ventilation mode. Therefore, BiPAP is a universal ventilation mode, which reduces the numerous ventilatory modes, during the course of ventilation therapy.

   Controlled ventilation
   ↕
   Augmented ventilation
   ↕
   Spontaneous ventilation

2. *Synchronization with the patient's breathing*

   The change over from the expiratory pressure level to inspiratory pressure level, and vice versa, is synchronized with the patient's breathing phase.

3. *Returns to spontaneous breathing more rapidly*

   As new clinical research has shown, this easy adaptation to the patient's spontaneous breathing requires less sedation, so that the patient returns to spontaneous breathing more rapidly.

SUMMARY

Initially, BiPAP was mainly used for weaning ICU patients who were on ventilatory support. Now a very large proportion (roughly 90%) of patients requiring mechanical ventilation are ventilated with BiPAP irrespective of severity of pulmonary failure and the severity of pulmonary disease, provided this is feasible for the ventilator. Even under the most invasive IRV ventilation there is, therefore, no need to suppress the patient's spontaneous breathing by deep sedation and relaxation. Thus, the introduction of BIPAP has significantly reduced the need for relaxants and sedatives.

This classic form of respiratory therapy for a ventilated patient includes all known ventilation modes, from controlled ventilation (IPPV) to step by step reduction of mandatory ventilation (SIMV), and from pressure-supported ventilation (ASB) to machine-applied spontaneous breathing (CPAP). With BiPAP all these respiratory therapies are possible in one mode. The switch over from low to high-pressure level is equivalent to pressure-controlled ventilation, and spontaneous breathing is possible at each pressure level.

Bi-vent/Bi-level – Pressure support to both levels (Fig. 32.16)

Bi-vent mode of ventilation is exactly like BiPAP, except Bi-vent mode also gives additional pressure support to spontaneous breath at also upper levels.

Bi-level is a combination of APRV and BiPAP; it mixes spontaneous and mandatory breath types. The mandatory breaths are pressure controlled and spontaneous breaths can be pressure supported. In Bi-level the ventilator cycling between the two pressure levels can be synchronized with the patient, to prevent a cycle to low pressure just as a patient take breath. Spontaneous breaths can be pressure supported at the high and low-pressure levels.

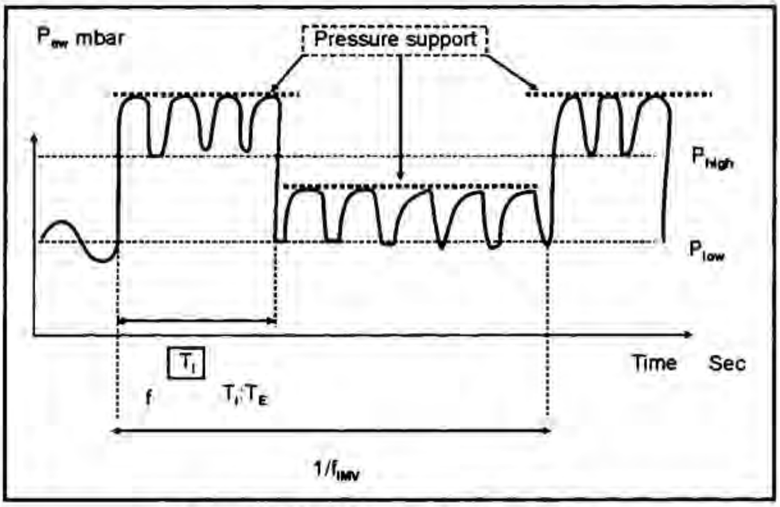


Figure 32.16: Bi-Vent mode of ventilation

The advantage of this method of ventilation is the decreased level of sedation required to facilitate patient acceptance of the ventilator and the concept of a single modality to ventilate the patient. Bi-level can offer full ventilator support and can then be weaned off, as patient ventilator needs resolve. If the patient's gas exchange deteriorates subsequently, it is possible to increase the "invasiveness" of ventilation.

CHAPTER

33

Continuous Positive Airway Pressure and Extrinsic Positive End-expiratory Pressure

CPAP/PEEP is not a ventilation mode but an adjunctive treatment or a technique of respiratory therapy which can be applied to all forms of mechanical ventilation controlled, assisted and to spontaneously breathing patients.

CPAP/PEEP (FIG. 33.1)

In CPAP/PEEP airway pressure is maintained above atmospheric pressure, throughout the respiratory cycle. When positive airway pressure

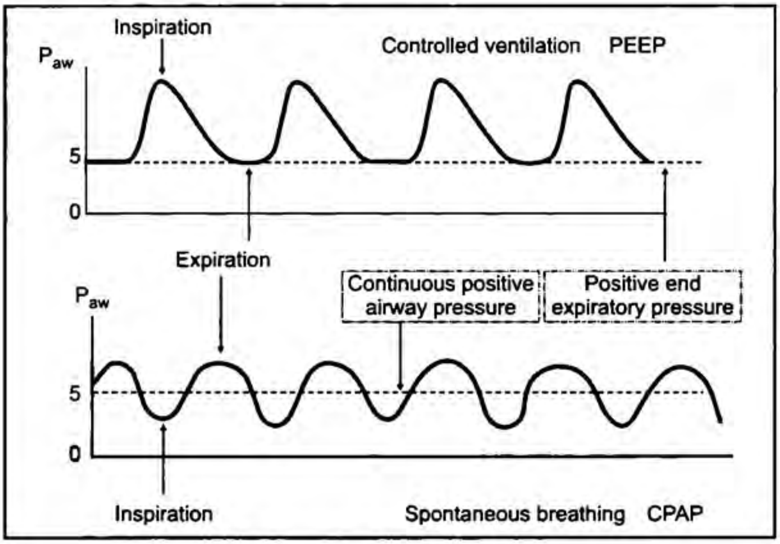


Figure 33.1: PEEP/CPAP

is applied during a spontaneous breath, it is called CPAP (continuous positive airway pressure) and during mechanical breath (assist or control), it is called PEEP (positive end-expiratory pressure).

The principle difference between PEEP and CPAP can be seen during inspiration. During controlled ventilation + PEEP, the pressure gradient responsible for inspiration is generated by a mechanical ventilator and intrapleural pressure increases. Whereas during spontaneous breathing with CPAP, the pressure gradient required for inspiration is generated by the respiratory muscle and intrapleural pressure decreases. The patient must have adequate lung functions that can sustain "eucapnic ventilation" before application of CPAP.

Pressure Preset Mode

When PEEP is applied to a pressure preset mode, actual pressure applied during inspiration is the sum of inspiratory and expiratory pressures. When the level of the PEEP is high, peak inspiratory pressure may be limited to prevent it from reaching dangerous levels that contribute to air leaks and barotrauma.

BiPAP/Bi-Level Mode

Expiratory positive airway pressure (EPAP) and inspiratory airway pressure (IPAP) are applied during BiPAP or Bi-level mode. In this mode IPAP indicates the total pressure applied (inclusive of EPAP) during inspiration.

FACTORS ESSENTIAL FOR KEEPING THE SMALL AIRWAYS OPEN

Alveolar Pressure – (P_{alv})

The alveolar pressure (P_{alv}) is the sum of the intrapleural pressure (P_{ple}) and the elastic recoil pressure (elastance) of the lungs.

$$P_{alv} = P_{ple} + \text{Elastic recoil pressure (Elastance)}$$

The mean intrapleural pressure (pressure within the pleural space) is exerted on the total lung including the airways.

Alveolar Distending Pressure

The thorax via parietal pleura and lungs via visceral pleura are mechanically united elastic structures with different resting states. With intact pleura, the thorax and lungs are mechanically interdependent. At the end of a normal exhalation, the respiratory muscles are relaxed, the tendency of the lungs to recoil inward and that of the thorax to recoil outward is balanced (i.e. the elastic recoil forces of lung and thorax are equal and opposite).

Normally, at the end of expiration the alveolar end-expiratory pressure equilibrates with the atmospheric pressure (i.e. zero pressure) and at that time the mean pleural pressure is approximately 5 cm H_2O less than atmospheric pressure (–5 cm H_2O) and the lungs are at their normal FRC.

Under these conditions, the normal alveolar distending pressure is 5 cm H_2O (alveolar pressure minus pleural pressure), which is sufficient to overcome the elastic recoil of the alveolar wall and to maintain a normal end-expiratory alveolar volume.

$$\begin{aligned}\text{Alveolar distending pressure} &= \text{Alveolar pressure - Pleural pressure}\\ &= 0 - -5\\ &= 5 \text{ cm } H_2O\end{aligned}$$

When the balance is upset (acute lung injury), the recoil forces are increased and FRC decreases.

Elastic Recoil of the Lungs (Figs 33.2 and 33.3)

During inspiration, elongation of the elastic pulmonary fibres increases the elastic recoil (comparable with a coiled spring). Elastic recoil of the lung is maximum at the height of inspiration and minimum at FRC.

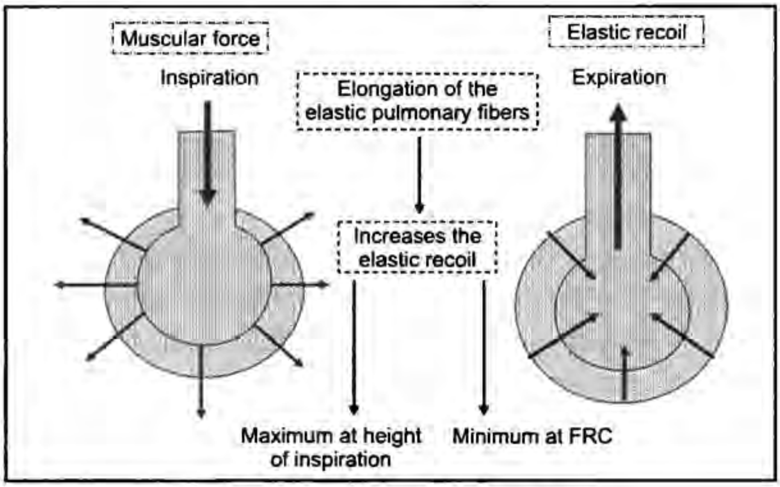

Muscular force
Inspiration
Elastic recoil
Expiration
Elongation of the elastic pulmonary fibers
Increases the elastic recoil
Maximum at height of inspiration
Minimum at FRC

Figure 33.2: Elastic recoil of the lungs

During inspiration, bronchioles are stretched by the stronger radial pull and flow resistance of bronchi falls. With expiration the elastic recoil decreases, the bronchi become narrower and the flow resistance increases.

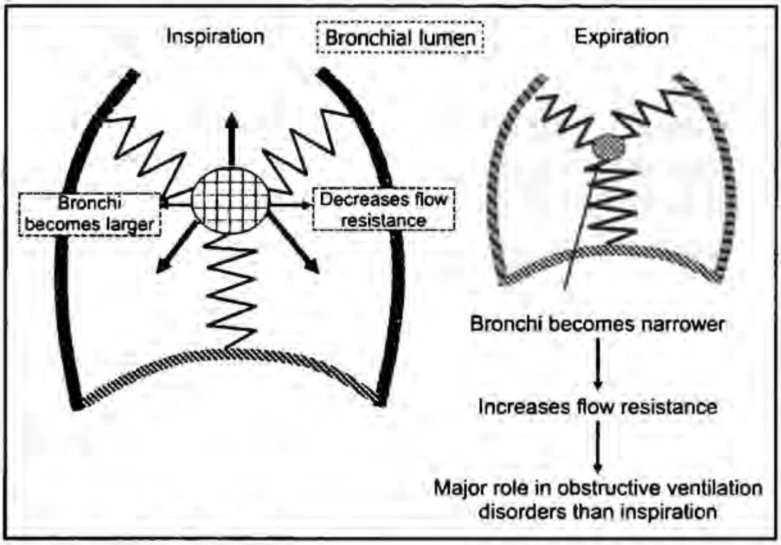

Inspiration
Bronchial lumen
Expiration
Bronchi becomes larger
Decreases flow resistance
Bronchi becomes narrower
Increases flow resistance
Major role in obstructive ventilation disorders than inspiration

Figure 33.3: Changes in bronchial lumen due to inspiration and expiration

That is why expiration always plays a major role in obstructive ventilation disorders than inspiration.

Decreased Elastic Recoil – Obstructive Ventilation Disorders

In emphysema the recoil of the lung is reduced and therefore the compliance is increased due to the loss of pulmonary parenchyma.

Increased Elastic Recoil – Restrictive Ventilation Disorder

When the balance of recoil forces (lung and chest) is upset, increase in elastance of the lungs produces reduction in respiratory system compliance and the reduction in FRC. It also increases airway resistance due to reduction in radial traction of peripheral airways. Reduced compliance is also referred to as a restrictive ventilation disorder.

There are various causes which are primarily responsible for the increase in the recoil forces of the lung (elastance), decreasing lung compliance such as:

- *Alveolar collapse – atelectasis*
  Loss of elasticity of the pulmonary fibre structures - pulmonary fibrosis
- *Reduction of alveolar size caused by hydrostatic compression* and the intrapulmonary fluid content - pulmonary oedema
- *Reduced surface tension due to damage to surfactant*
  Acute lung injury

EFFECTS OF DECREASED COMPLIANCE (FIG. 33.4)

Decreased compliance will increase elastic recoil, decreasing alveolar volume. If the lung compliance continues to deteriorate, the elastic recoil forces can become great enough to completely overcome the normal alveolar distending pressure, thus resulting in alveolar collapse and the creation of intrapulmonary shunting.

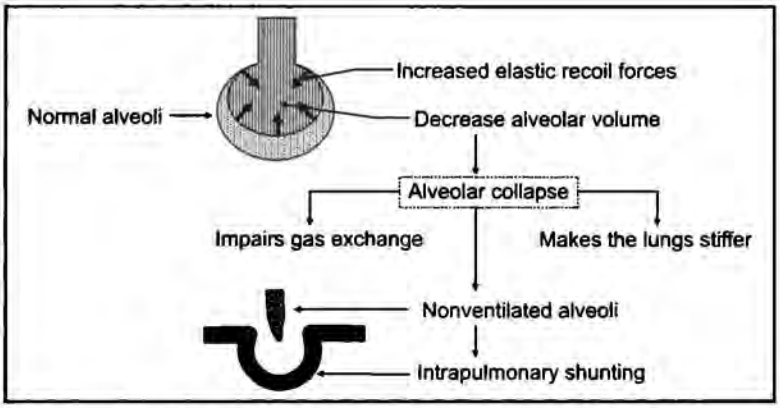


Figure 33.4: Effects of decreased compliance

The distal airspace tend to collapse at the end of expiration, and this tendency is exaggerated when the lungs are stiff (e.g., in ARDS). Alveolar collapse impairs gas exchange and makes the lungs stiffer. The reduction of respiratory system compliance reduces the expiratory time constant.

COMPENSATORY MECHANISMS TO INCREASE RESIDUAL LUNG VOLUME

Tachypnoea (Fig. 33.5)

Pulmonary baroreceptors are stimulated by an increase in the recoil forces of the lung and also to an abnormally low FRC. Stimulation of pulmonary baroreceptors sends impulses to the respiratory control center, which in turn produces tachypnoea and glottic narrowing during expiration.

Baroreceptors induced tachypnea is a compensatory mechanism to increase residual lung volume; it often precedes gas exchange abnormalities (i.e. arterial hypoxaemia).

Expiratory Grunting

Glottic narrowing (expiratory grunting) is another physiologic compensation to brake gas flow, thus increasing the expiratory time constant and residual lung volume in patients without an artificial airway.

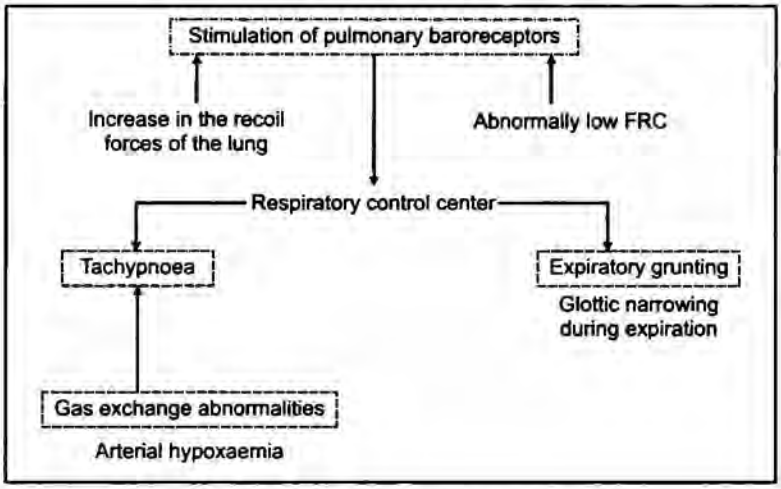

Stimulation of pulmonary baroreceptors
Increase in the recoil forces of the lung
Abnormally low FRC
Respiratory control center
Tachypnoea
Expiratory grunting
Glottic narrowing during expiration
Gas exchange abnormalities
Arterial hypoxaemia

Figure 33.5: Mechanism producing tachypnoea

Expiratory grunting does not effectively prevent, but merely delays development of expiratory atelectasis.

EFFECTS OF EXTERNAL PEEP ON VARIOUS SYSTEMS

- Pulmonary effects
- Cardiovascular effects
- Noncardiorespiratory effects of PEEP
  - Bronchial circulation
  - Renal function
  - Barotrauma
  - Auto-PEEP

Pulmonary Effects

Arterial blood gas tensions represent the mean partial pressure of carbon dioxide $PaCO_2$ and oxygen PaO_2 in perfused and ventilated alveoli. Because the majority of gas exchange occurs during expiration (in mechanically ventilated patient), effective improvement must occur primarily during expiration. There are two universal pulmonary effects of external PEEP, i.e. redistribution of extravascular water and increased functional residual capacity which are beneficial for gas exchange during expiration.

1. *Redistribution of extravascular water (Fig. 33.6)*
   PEEP therapy does not decrease total lung water. The redistribution of extravascular lung water, reducing intrapulmonary shunt, is a major mechanism by which PEEP improves lung function. The application

of PEEP to the edematous lung decreases intra-alveolar fluid volume and facilitates the movement of water from the less compliant interstitial spaces (between the alveolar epithelium and pulmonary capillary endothelium) where gas exchange occurs, to the more compliant interstitial spaces (peribronchial and hilar regions).

This redistribution helps to reinflate previously flooded and collapsed airspaces, without decreasing lung oedema.

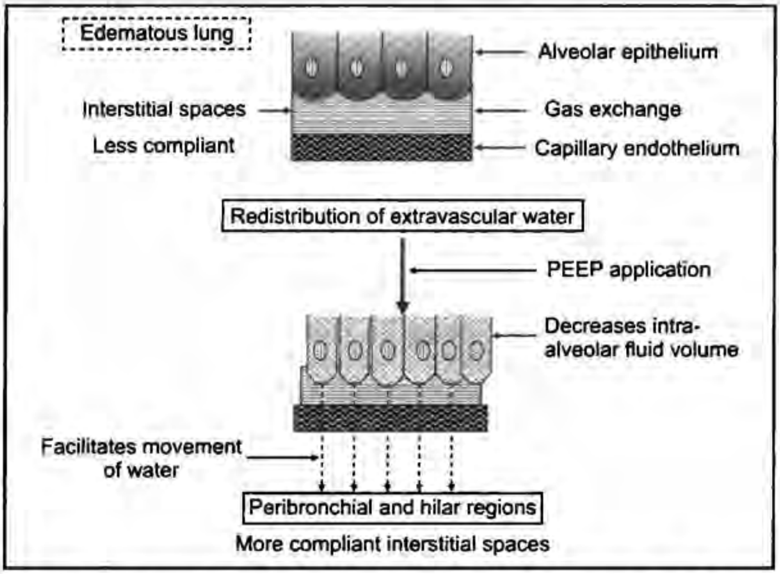

Edematous lung
Alveolar epithelium
Interstitial spaces
Less compliant
Gas exchange
Capillary endothelium
Redistribution of extravascular water
PEEP application
Decreases intra-alveolar fluid volume
Facilitates movement of water
Peribronchial and hilar regions
More compliant interstitial spaces

Figure 33.6: Re-distribution of extravascular water (For colour version *see* plate 15)

2. *Increased functional residual capacity (FRC)*
   IPPV is intended to replace in part or completely, the function of the respiratory muscles, to improve alveolar hypoventilation, correcting hypoxaemia. However, hypoxaemia due to intrapulmonary shunt or venous admixture requires recruitment of collapsed alveoli.

PEEP Results in an Increased FRC by Two Distinct Mechanisms

- *Increasing the volume of patent alveoli* (Fig. 33.7)
  This usually happens with PEEP levels below about 1O cm H_20.
- *Opening up of previously closed alveoli*
  Mechanical ventilation usually is not effective in improving oxygenation in patients with intrapulmonary shunt. When inspiration terminates and expiration begins, alveolar collapse recurs. Pressure at which the alveoli collapse is called *alveolar closing pressure;* this is due to increased elastic recoil pressure.

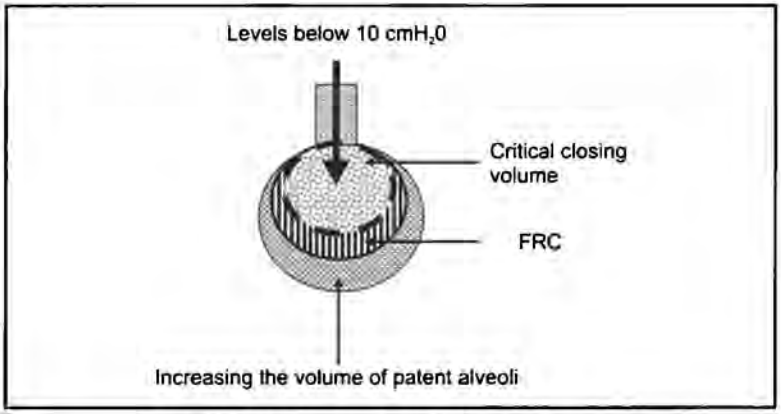


Figure 33.7: Increasing the volume of patent alveoli

Alveolar opening pressure is pressure necessary to open collapsed alveoli (recruitment). The alveolar opening pressure is always higher than the alveolar closing pressure. During inspiration, recruitable alveoli are inflated when sufficient 'alveolar opening pressure or *recruitment pressure*' is applied (Fig. 33.8)

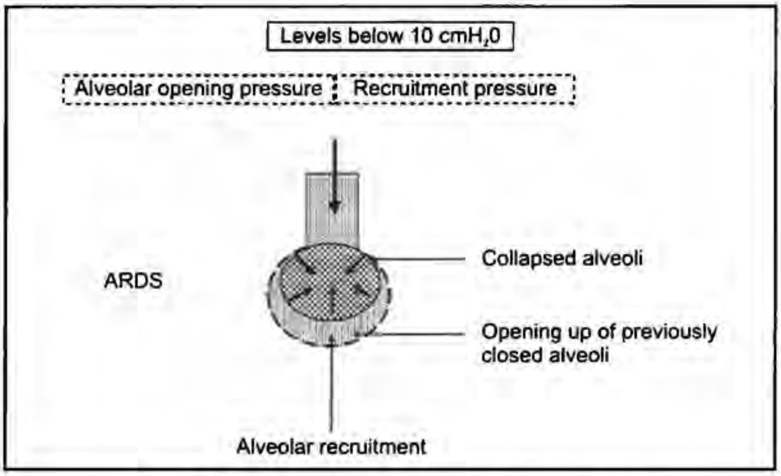


Figure 33.8: Opening up of previously closed alveoli

Once opened, alveolar patency can be sustained by applying end-expiratory pressure, which is equal to the *'alveolar distending pressure or de-recruitment pressure'*. De-crecruitment is prevented with less pressure than was required for recruitment (Fig. 33.9)

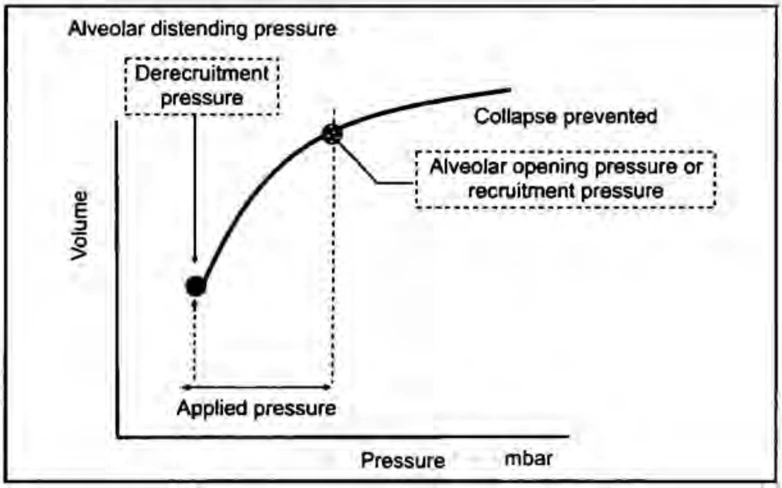

Alveolar distending pressure
Derecruitment pressure
Collapse prevented
Alveolar opening pressure or recruitment pressure
Volume
Applied pressure
Pressure mbar

Figure 33.9: Alveolar recruitment during inspiration and de-recruitment during expiration

This phenomenon is called hysteresis and is characteristic of elastic structures, where recoil forces are less than expansion forces.

PEEP increases the alveolar distending pressure and acts as a counterforce which is sufficient to maintain a normal end-expiratory alveolar volume to overcome the elastic recoil of the alveolar wall and therefore re-expands or "recruits" these collapsed alveoli and maintains inflation during exhalation.

Once "recruitment" of these alveoli has occurred, PEEP lowers the alveolar distending pressure and facilitates gas diffusion and oxygenation (Fig. 33.10)

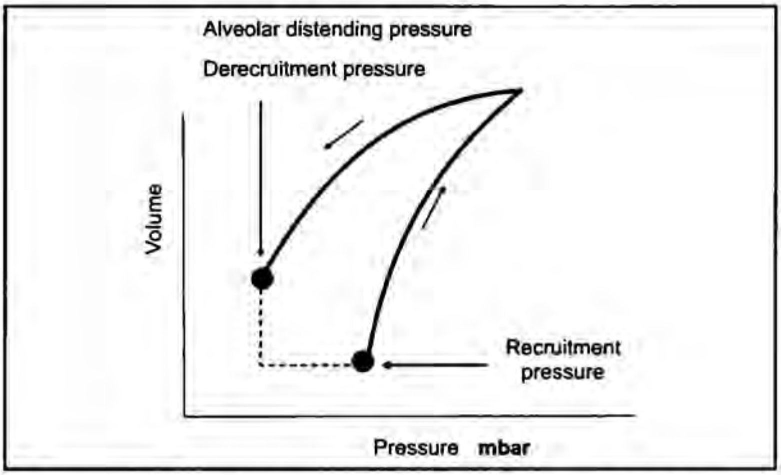


Figure 33.10: Alveolar recruitment during inspiration and prevention of derecruitment by the application of PEEP

Higher levels of PEEP are generally responsible for alveolar recruitment, i.e. opening up of previously closed alveoli. Application of PEEP will prevent 'de-recruitment' and also will maintain alveolar patency throughout the ventilator cycle (Fig. 33.11)

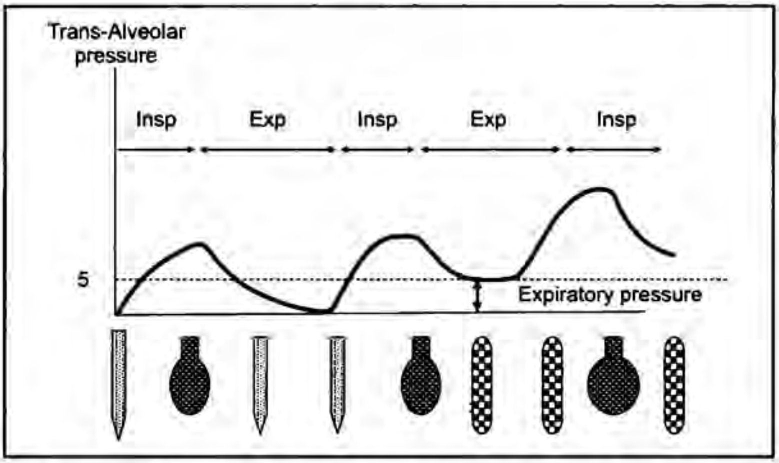


Figure 33.11: Action of PEEP to prevent 'de-recruitment' and maintain alveolar patency throughout the ventilator cycle

Prevention of alveolar collapse tends to increase respiratory system compliance making lungs less stiff (i.e. decreases lung recoil forces), decreases recoil rate (i.e. increases expiratory time constant), reduces baroreceptors mediated tachypnea and improves arterial oxygenation at a given level of oxygen concentration (i.e. oxygenation tends to be proportional to the gas exchanging surface area of the lungs). Alveolar patency decreases intrapulmonary shunt.

This improved gas exchange raises the arterial PaO_2, which allows the inspired oxygen (FiO_2) to be reduced to less toxic levels. This latter effect (decrease to less toxic inhaled O_2 concentration) is one of the major indications for extrinsic PEEP.

PEEP- increases the alveolar end-expiratory pressure

↓

Increases alveolar distending pressure

↓

Increases FRC by alveolar recruitment/prevention of de-recruitment

↓

Improves ventilation

↓

Increases V_A/Q, improves oxygenation, decreases work of breathing

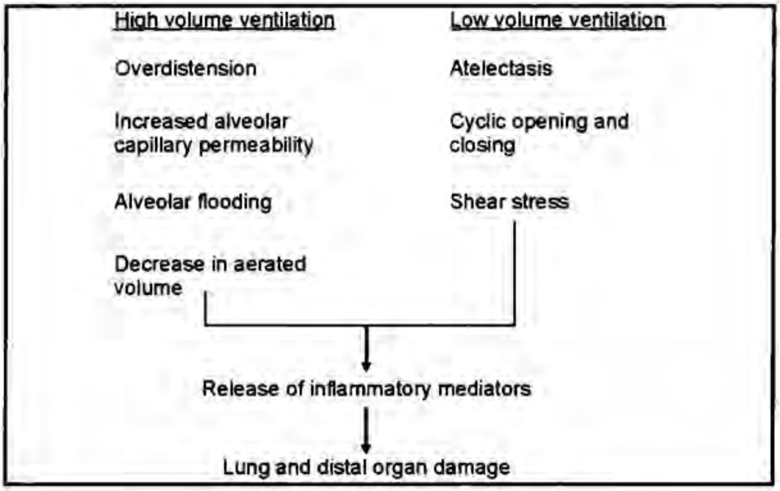
↓

Decrease to less toxic inhaled O_2 concentration

When the elastic recoil force of the lungs reduces, the efficiency of spontaneous breathing usually improves, thus reducing the requirement for mechanical ventilation. Indeed alveoli that are recruited and de-recruited during tidal ventilation may eventually become unrecruitable because the surfactant lining is damaged by rhythmic reopening and collapsing. Moreover prevention of rhythmic alveolar reopening and collapse may preserve surfactant function and decrease the shear forces responsible for lung parenchymal damage causing barotrauma.

End-expiratory volume is increased by an amount equal to the product of end-expiratory pressure and respiratory system compliance. Because alveoli in patients with acute lung injury tend to be either healthy and recruitable or diseased and not recruitable, the effect of end-expiratory pressure on FRC is variable.

End-expiratory pressure tends to cause relative hyperinflation and a decrease in respiratory system compliance of healthy alveoli. End-expiratory pressure applied, equal to alveolar de-recruitment pressure, will prevent expiratory collapse, and improve respiratory system compliance in recruited alveoli. Thus, end-expiratory pressure has conflicting effects on regional alveolar volume and respiratory system compliance.


High volume ventilation
Low volume ventilation
Overdistension
Atelectasis
Increased alveolar capillary permeability
Cyclic opening and closing
Alveolar flooding
Shear stress
Decrease in aerated volume
Release of inflammatory mediators
Lung and distal organ damage

Overdistension of the alveolar septa involves a loss of elasticity. There is danger of structural damage to the alveoli (baro/volutrauma) and decrease in perfusion due to capillary compression.

Cardiovascular Effects

The respiratory system affects cardiovascular function primarily by impairing venous return. When cardiac function is normal, venous return is the predominant determinant of cardiac output. The rate at which systemic venous blood returns to thoracic veins depends on the transthoracic vascular pressure gradient (i.e. extrathoracic pressure minus intrathoracic pressure). The transthoracic pressure gradient is determined largely by intrapleural pressure, which is normally subatmospheric.

Magnification of Cardiac Effect by PPV with PEEP (Fig. 33.12)

Because PEEP displaces the entire positive-pressure waveform upward, the effects of positive pressure ventilation on cardiac performance is magnified.

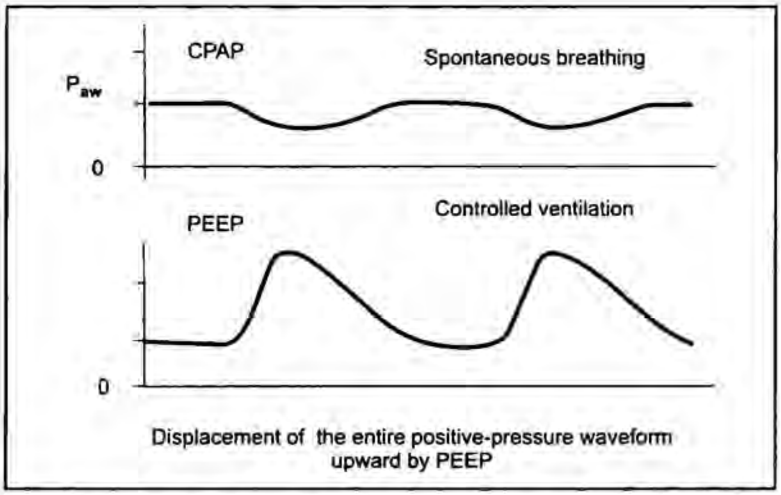


Figure 33.12: Magnification of cardiac effect by PPV with PEEP

Thus, the tendency for PPV to reduce cardiac filling and cardiac output is much greater with PEEP; this tendency is magnified even further by hypovolemia and cardiac dysfunction.

Clinical Significance of Mean Intrathoracic Pressure

The tendency for PEEP ventilation to reduce cardiac output is not a function of the PEEP level, but is a function of the PEEP induced increase in mean intrathoracic pressure. Even low levels of PEEP can be harmful for the cardiac output if the mean intrathoracic pressure is high.

A diminished cardiac output with PEEP is attributable to at least 3 mechanisms, i.e. decreased venous return to the right heart, PEEP increases right ventricular afterload, transmission of increased alveolar pressure.

1. *Factors influencing decreased venous return to the right heart*

   Assuming a normal intravascular volume, venous return to the right atrium is influenced by the difference in the central venous pressure and the negative pleural pressure that surrounds the heart. Intrapleural pressure is created by two opposing forces: - lung recoil (visceral pleura) and chest wall retraction (parietal pleura). Any changes in either of these forces will alter intrapleural pressure and the transthoracic vascular pressure gradient.

   a. ***Types of breathing***

   - *Spontaneous breathing*
     - Inspiration

       As the volume of thorax increases during spontaneous inspiration, intrapleural pressure decreases from an average of 5 cm H_2O to 10 cm H_2O below atmospheric pressure. The vena caval, pulmonary arterial and aortic pressure decreases immediately because the vessels dilate when the extramural pressure decreases. Both the cardiac output and the systemic arterial pressure decreases because of the temporary increase in pulmonary vascular capacitance. As the transthoracic vascular pressure gradient increases, venous return and right ventricular stroke volume increases. At the end-inspiration the pulmonary arterial pressure and aortic pressure, as well as the cardiac output, diminishes because increased venous blood flow fills the expanded pulmonary vascular capacitance.
     - Expiration

       During spontaneous exhalation, intrapleural pressure and pulmonary artery blood flow return to baseline, the cardiac output and systemic arterial pressure increases as pulmonary vascular capacitance diminishes. Thus, cardiac output and systemic arterial pressure fluctuates with breathing patterns, reflecting phasic alteration in blood flow into and out of the thorax.

   - *Mechanical ventilation*

     This process is reversed during mechanical ventilation. Intrapleural pressure increases when mechanical ventilation is initiated, and after the application of end-expiratory pressure. thereby decreasing transthoracic vascular pressure gradient, venous inflow, right ventricular stroke volume, left ventricular stroke volume and cardiac output. The fractional transmission of the end-expiratory pressure to the pleura depends on the mechanical properties of the lung and chest wall.

When end-expiratory pressure is employed with spontaneous ventilation (CPAP), it affects cardiovascular function differently than when it is provided with mechanical ventilation (CMV +PEEP).

- *Increase in the mean airway pressure*

  A significant increase in the mean airway pressure is more likely to increase pleural pressures sufficiently to decrease venous return. Since PEEP increases both peak inspiratory pressures and mean airway pressures, it has the potential to decrease venous return and cardiac output. During PEEP, the pleural pressure becomes less negative and the pressure gradient between the central venous drainage and the right atrium will decrease resulting in a decreased venous return. This results from increased intrathoracic pressure. The increase in mean airway pressure associated with PEEP increases pleural and pericardial pressure, which may significantly decrease the end diastolic volume and stroke volume of both ventricles. This in turn results in a decreased cardiac output and hypotension. This reduced stroke volume, can be restored by preload augmentation. When ventilating with PEEP, considerations must be given to decreased venous return, cardiac output, blood pressure, and organ perfusion.

  During spontaneous exhalation and during mechanical ventilation with the same end-expiratory level, intrapleural pressure and venous return are similar. Therefore, at a given level of end-expiratory pressure, the most important determinant of mean airway pressure, intrapleural pressure and transthoracic vascular pressure gradient is the inspiratory airway pressure pattern.

b. ***Pulmonary vascular resistance***

End-expiratory pressure may reduce or increase pulmonary vascular resistance. Principle respiratory factors that affect pulmonary vascular resistance, pulmonary perfusion and blood flow distribution are airway pressure, FRC and hypoxic pulmonary vasoconstriction. When FRC is normal, pulmonary vascular resistance is minimal. Changes in lung volume, above or below normal, FRC increases pulmonary vascular resistance. Therefore, FRC should be normalized whenever possible. End-expiratory pressure is titrated to restore normal FRC, which also recruits perfused, but non-ventilated lung units, thus improving PaO_2 and relieving hypoxic pulmonary vasoconstriction.

2. *PEEP increases right ventricular afterload*:
   PEEP increases pulmonary vascular resistance. At higher levels of PEEP, ventricular septal shifting (Bernheim effect) towards the left may occur, limiting distensibility of LV. In patients with LV dysfunction and elevated filling pressures, PEEP may improve cardiac function by improvement in coronary arterial oxygen content by reduction of venous return.
3. *Transmission of increased alveolar pressure:*
   Normally, more than 1/2 of the elevated alveolar pressure is dissipated by the lungs elastic forces, however, poorly compliant lung less than 1/2 of elevated alveolar pressure is transmitted conversely extremely compliant lungs transmit a greater degree of pressure, which can lead to hemodynamic consequences.

Precautions while Applying PEEP – Check BP

It is vital to closely monitor the patient receiving PEEP therapy for any drop in blood pressure, especially when PEEP is either first applied or increased to high levels. If PEEP decreases the blood pressure, first be sure that the patient is not hypovolemic. If the blood volume is adequate then the PEEP should be decreased until an adequate blood pressure is re-established. A given amount of PEEP does not impede venous return to the same degree in different patients.

Low lung compliance:
If a patient has extremely low lung compliance, the airway pressure is less readily transmitted into the pleural space. The low lung compliance shields the pleural space from the effects of increased alveolar pressure. Patients with ARDS usually have low lung compliance, often requiring very high PEEP levels. However, despite high PEEP levels haemodynamic instability is seldom a problem, due to dampening effect unless the patient has a pre-existing cardiovascular disease.

Normal or elevated lung compliance:
In contrast, if a patient has normal or elevated lung compliance, the increased alveolar pressure due to the PEEP will be more readily transmitted into the pleural space. In other words PEEP therapy in patients with normal or high lung compliance is more likely produce an elevated pleural pressure and therefore a decreased venous return.

Mean airway pressure:
Mean airway pressure is influenced by inspiratory time, respiratory rate, peak inspiratory pressure and PEEP. These four parameters should be kept minimum in order to maintain the MAWP at the lowest level possible. Regardless of the mode of ventilation selected on the ventilator, a higher MAWP usually results in a lower cardiac output.

In comparing CPAP and PEEP, PEEP reduces the cardiac output more severely as it causes a higher MAWP (Fig. 33.13).

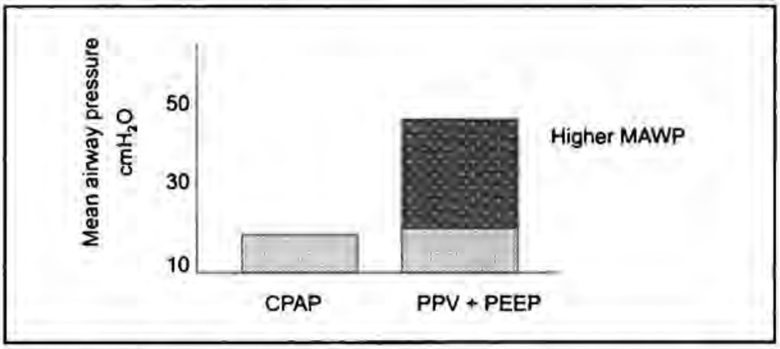


Figure 33.13: PEEP reduces the cardiac output more severely

This is because PEEP is used in addition to PPV, whereas CPAP is used in spontaneously breathing patients.

Clinical Significance of Cardiac Output and Determinants of Systemic Oxygen Delivery

The tendency for PEEP to reduce the cardiac output is an important consideration, as demonstrated by the determinants of systemic oxygen delivery (DO_2).

$$DO_2 = CO \times 1.36 \times Hb \times SaO_2$$

Thus, the effect of PEEP on cardiac output will determine whether a PEEP-induced increase in arterial oxygenation (SaO_2) is associated with a similar increase in systemic oxygenation. When PEEP does not change the cardiac output, the increase in arterial O_2 saturation is associated with an increase in systemic O_2 delivery. However, when PEEP reduces the cardiac output, the increase in arterial oxygenation is associated with a decrease in systemic oxygenation. The point at which PEEP improves systemic oxygen transport is sometimes called best PEEP.

Precautions

An increase in arterial oxygenation should never be used as the end-point of PEEP ventilation; rather the cardiac output must be monitored when PEEP is applied. There is a waiting period of at least 15 minutes after PEEP is applied before the effects on cardiac output are fully expressed. If cardiac output monitoring is not available, the venous oxygen saturation can be useful. A decrease in venous O_2 saturation after PEEP can be used as evidence for a PEEP induced decrease in cardiac output.

NON-CARDIORESPIRATORY EFFECTS OF PEEP

1. *Bronchial circulation*

   PEEP could interfere with the bronchial circulation by decreasing tota cardiac output, increasing resistance in the pulmonary circulation, compressing the bronchial vessels perfusing the airways.

2. *Effects on kidney:*

   Mechanical ventilation and PEEP, decreases means renal arter perfusion pressure; redistribute RBF from the cortical to medullary regions, which may account for increased sodium retention. PEEP decreases atrial natriuretic peptide production by mechanically compressing the atrium or by decreasing right atrial stretch due to decreased venous return. PEEP may cause inhibition of aldosterone production, and increase antidiuretic hormone. Low dose dopamine infusion and the intravascular hydration improve renal function. CPAP is less detrimental than PEEP.

3. *Effects on brain*

   Because end-expiratory pressure reduces the transthoracic vascular pressure gradient, venous return is impeded. This impedes venous return from the head, may cause increase in intracranial pressure, a decrease in cerebral blood flow, and may reduce cerebral perfusion pressure. Spontaneous ventilation and/or elevation of the head 10 to 15 degrees may increase the transthoracic vascular pressure gradient decrease intracranial pressure and improve cerebral perfusion.

MECHANISM OF APPLICATION OF PEEP/CPAP (FIG. 33.14)

PEEP can be applied placing a pressure-limiting valve in the expiratory limb of the ventilatory circuit. This valve exerts a backpressure and exhalation proceeds until this back pressure is reached, whereupon flow ceases.

GOALS OF PEEP

- *Maintaining stability of alveolar segments*
  - Increasing FRC above closing volume to prevent alveolar collapse
  - PEEP opens up the alveoli and keeps those alveoli open.
- *Improvement of oxygenation – increase in PaO_2*
  - Increasing the FRC – Increasing the gas exchange area
  - Reopening atelectatic lung area – alveolar recruitment
  - Reducing the right to left shunt
  - Avoiding end-expiratory alveolar collapse
  - Improving the ventilation/perfusion ratio

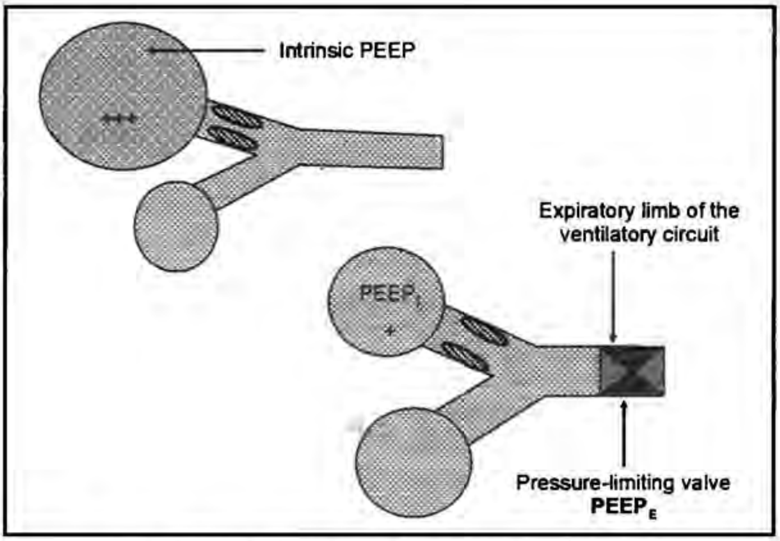


Figure 33.14: Mechanism of applications of PEEP/CPAP

- *Reduction in work of breathing* – by improving lung compliance
- *Reduction in inspired oxygen concentration to 'nontoxic' levels* (usually less than 50%) and maintenance of PaO_2 > 60 mm Hg or SaO_2 >90%.

INDICATIONS FOR PEEP

Allows the Inhaled Oxygen to be Reduced to Less Toxic Levels

A minor role is played by ventilation/perfusion mismatch in hypoxaemic respiratory failure which is caused by cardiogenic pulmonary oedema, ARDS, unilateral pneumonia. Refractory hypoxaemia in this patient is due to intractable shunt (primary indication for PEEP), caused by interstitial and alveolar oedema, small airway closure, atelectasis, reduction of FRC, low V/Q (alveoli are perfused but not ventilated, e.g. mucus or atelectasis). In this type of pathology, if high a FiO_2 is given, it may decrease lung volume further, by promoting alveolar denitrogenation and reabsorption atelectasis.

Refractory hypoxemia is defined as hypoxaemia that responds poorly to moderate to high levels of oxygen. A helpful clinical guideline for refractory hypoxemia is when the patient's PaO_2 is 60 mm Hg or less at FiO_2 of 50 % or more. In patients who require a toxic level of inhaled oxygen (FiO_2 greater than 0.60) to maintain adequate oxygenation, the addition of PEEP can increase arterial and systemic oxygenation and allow the inhaled oxygen to be reduced to less toxic levels.

Low–Volume Ventilation

PEEP is recommended for volume-cycled ventilation with low tidal volumes (5-10 ml/kg.) The PEEP is meant to prevent repetitive opening and closing of small airways, which is believed to be a source of further lung injury. The level of PEEP should be above the inflection point of the pressure-volume curve (the point of airway closure) but in the absence of such curve, a PEEP level of 10 cm H_2O may be satisfactory.

USE OF PEEP IN CLINICAL SETTINGS

ARDS/Acute Lung Injury

Goals

- Improve gas exchange
- Increase the amount of aerated lung volume
- Reduce intrapulmonary shunt
- Improve respiratory mechanics.

Pathophysiology in ARDS

Impairment of gas exchange:

Severe impairment of gas exchange occurs in ARDS because of increased intrapulmonary shunt resulting from flooded and collapsed alveoli which is characteristic of ARDS and unilateral pneumonia. Surfactant abnormalities, increased airway resistance, inflammatory proteins and cell accumulating within alveoli contribute to intrapulmonary shunt. Interstitial oedema, deposition of the cells and connective tissue, in the interalveolar septa, further contribute to gas exchange impairment. In ARDS, the altered blood flow distribution resulting from widespread involvement of the pulmonary vasculature and impaired hypoxic vasoconstriction response contribute to worsening of gas exchange.

Increases amount of non-aerated lung volume:

Reabsorption atelectasis, ventilation occurring at low lung volumes, and impairment of hypoxic vasoconstriction response increases amount of non-aerated lung volume.

Compression atelectasis:

There is a definite correlation between the extent of lung densities found mainly in the dependent regions on CT scan and deterioration in arterial blood gases. Oedema may increase total mass of the lungs (up to more than twice that of normal lungs). Consequently, the dependent zone collapses progressively under the weight of superimposed lung (compression atelectasis) and aerated lung decreases (concept of baby

Increases the work of breathing leading to fatigue:

A severely diminished FRC and reduced lung compliance greatly increases the alveolar opening pressure. If the patient is breathing spontaneously, decreased lung compliance always increases the work of breathing and if severe enough can lead to fatigue of the respiratory muscles and ventilatory failure. Since PEEP increases the FRC this pulmonary impairment may be prevented or improved by early application of PEEP therapy.

How PEEP Helps in ARDS (Fig. 33.15)

Although an increase in lung volume is the main mechanism for PEEP induced changes in oxygenation, a small decrease in cardiac output also reduces intrapulmonary shunt and improves PaO_2.

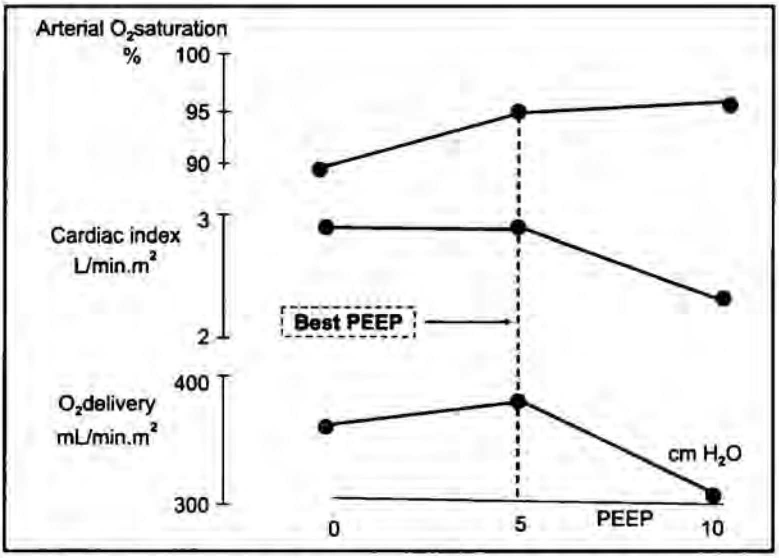


Figure 33.15: Effects on incremental PEEP on arterial and systemic oxygenation in a patients with severe ARDS

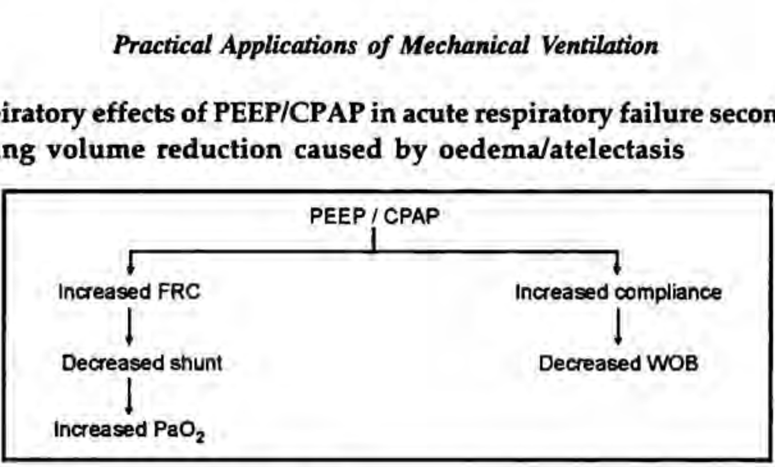
Promotes alveolar recruitment:

PEEP promotes alveolar recruitment and increases aerated lung volume, improving arterial oxygenation.

A redistribution of alveolar oedema:

A redistribution of alveolar oedema from the interstitial spaces improves gas exchange. By recruiting non-aerated alveoli and stabilizing airways, PEEP also affects the regional distribution of tidal ventilation. When the predominant effect of PEEP is recruitment, alveolar ventilation is expected to become homogenous, particularly in the dependent zone.

Respiratory effects of PEEP/CPAP in acute respiratory failure secondary to lung volume reduction caused by oedema/atelectasis



How to Apply PEEP in ARDS

PEEP has become established therapy for patients with ARDS or with flow limitation.

Pioneering studies have shown that application of PEEP in acute lung injury patients undergoing mechanical ventilation increases oxygenation, FRC and respiratory compliance at moderate levels; although at high levels compliance may decrease.

ARDS Net Trial

On the other hand, the ARDS Net trial is used as a protocol in which combinations of FiO_2 and PEEP were fixed, and adjusted to the arterial oxygenation goal, PaO_2 = 55-80 mm Hg or SpO_2 = 88-95%.

Use the following incremental FIO_2/PEEP combinations to maintain arterial oxygenation within the target range.

FiO_2	0.3	0.4	0.4	0.5	0.6	0.6	0.7	0.7	0.7	0.8	0.9	0.9	0.9	1
PEEP	5	5	8	8	10	10	10	12	14	14	14	16	18	20-24

Postoperative Hypoxaemia

Postoperative hypoxaemia is mainly caused by atelectasis. After various surgical procedures, mainly upper abdominal and thoracic, atelectasis develops in 30-50% of cases and leads to acute respiratory failure (ARF). However, only 8-10% of patients require intubation and mechanical ventilation.

CPAP is a simple, practical, and safe technique for managing postoperative hypoxaemia; if applied for a sufficient time. In cardiac and non-cardiac surgery, the application of CPAP (5-10 cm H_2O) with or without pressure support via face mask, reduces atelectasis and improves oxygenation, without increasing surgical complications such as anastomotic leak. By reducing atelectasis, CPAP may also decrease bacterial growth in the lung, prevent bacterial translocation into the blood stream and normalize alveolocapillary permeability.

COPD

COPD patients have abnormally increased respiratory system compliance (emphysema), giving rise to abnormally increased FRC and increased airway resistance (R_{aw} – bronchitis), leading to prolonged expiratory time constant.

Pathophysiology in COPD

Air trapping

Incomplete expiration rise to air trapping, which causes positive elastic recoil pressure at the end of expiration, hence the term occult pressure.

Increases inspiratory load for respiratory muscle

Air trapping in spontaneously breathing patients, requires that the respiratory muscles generate sufficient force, to oppose and overcome the positive recoil pressure, which will create a gradient, between the alveoli and airway opening to initiate airflow.

Air trapping therefore increases inspiratory load for respiratory muscle in spontaneously breathing patients or patients undergoing assisted CMV to trigger the ventilator.

Inspiratory load leads to a fatigue

The increase in respiratory muscle load and hyperinflation reduces the force generating capacity of the diaphragm. When the force-load balance is altered to a level that severe acidosis ensues, mechanical ventilation become necessary.

Diffusion impairment is not a major factor

In obstructive lung disease, diffusion impairment is not a major factor like acute lung injury but impaired gas exchange is caused by disturbances of regional ventilation and perfusion relationship, associated with alveolar hypoventilation. During an acute exacerbation of COPD, high V/Q areas predominates in the patients, mostly have the emphysematous changes with loss of blood flow, consequent to alveolar wall destruction. Low V/Q regions are seen mostly in patients with predominant obstructive component.

How External PEEP Helps in COPD

The ventilator acts as an accessory pump to reverse force and load imbalance. By reducing oxygen cost of breathing and correcting arterial blood gases, mechanical ventilation can improve respiratory muscle function (force). Respiratory assistance by the ventilator, however, does not reduce the inspiratory resistive load, consequent to airway resistance or the threshold load due to intrinsic PEEP.

Reduces the threshold load imposed on respiratory muscles
Application of external PEEP during assist control ventilation or CPAP in a spontaneously breathing patient counterbalances and reduces the inspiratory load imposed by air trapping. Thus, reducing the work of breathing, improving patient-ventilator interaction and reducing ineffective inspiratory efforts, external PEEP improves ventilatory efficiency.

Promote faster and more uniform lung emptying
The small airways tend to collapse, at the end of expiration in patients with obstructive lung diseases. Application of PEEP 5-10 cm H_2O in patients with COPD might partly decrease expiratory resistance, particularly at the end of expiration. This may keep the airways open, promote faster and more uniform lung emptying and reduce the tendency for air trapping. However, PEEP does not reduce hyperinflation and its effects on diaphragmatic force-generating capacity.

Effectiveness of non-invasive ventilation in acute exacerbation
CPAP is effective in reducing dyspnea, decreasing work of breathing and improving cardiac function. Most trials evaluated the effectiveness of non invasive ventilation, using PEEP of 4-5 cm H_2O in addition to pressure support, in patients with acute hypercapnia, secondary to COPD exacerbation.

Benefit to treat hypoxaemia is less
Unless hypoxaemia is caused by a large intrapulmonary shunt, the benefit is small and of little clinical significance and PEEP may have negative haemodynamic effects that worsen oxygen delivery. The balance between positive and negative effects depends on the level of PEEP applied.

Favourable Effects of Pharmacotherapy

Pharmacotherapy can have favourable effects on both load reduction and force restoration. Agents used should act on bronchial smooth muscle (beta agonist, anticholinergics) or inflammatory process (glucocorticoids and antibiotics). These agents decrease airway resistance, dynamic hyperinflation and intrinsic PEEP in ventilated patients. Glucocorticoids and antibiotics are commonly administered parenterally. In ventilated patients, bronchodilators are given more effectively and safely via a small volume nebulizer or a metered dose inhaler.

Acute Severe Asthma

Patients with acute severe asthma have high airway resistance, dynamic hyperinflation and intrinsic PEEP. Though some flow limitation is present, the main cause of dynamic hyperinflation and intrinsic PEEP is the prolonged expiratory time constant, secondary to high expiratory resistance. This increases respiratory muscle load and decreases force generation capacity.

Some alveolar units may be entirely occluded by mucous plugs and thus completely disconnected from the central airway. These patients are generally less responsive to external PEEP. In the absence of flow limitation, PEEP increases hyperinflation and alveolar pressure. Use of PEEP in patients receiving mechanical ventilation for acute severe asthma is often detrimental and thus not advisable. Very few data suggest that low level of non-invasive CPAP combined with pressure support in less severe and selected patients might be beneficial, although further evaluation is required.

Cardiogenic Pulmonary Oedema

CPAP should be considered first line of ventilator treatment, in severe cardiogenic pulmonary oedema. CPAP 10 cm H_2O is beneficial for achieving prompt physiologic improvement and a lower rate of endotracheal intubation in patients with cardiogenic pulmonary oedema. CPAP alone or in association with pressure support improves gas exchange by increasing aerated lung volume and improving cardiac output and ventilation perfusion distribution. Left ventricular failure causes an increase in left atrial and pulmonary venous pressure. Rise in pulmonary venous pressure, increases hydrostatic pressure and eventually the interstitial space is maximally expanded giving rise to flooding of alveoli. This decreases respiratory system compliance, FRC and expiratory time constant.

Patients with congestive cardiac failure may benefit from the haemodynamic consequences of end-expiratory pressure. In CCF, left ventricular contractility is reduced and relatively unresponsive to changes in preload. In these patients decreasing left ventricular afterload will improve cardiac output. Because increased pleural pressure reduces left ventricular afterload, end-expiratory pressure may improve cardiac performance and lung function in patients suffering from congestive cardiac failure. The application of PEEP to patients with either cardiogenic or noncardiogenic pulmonary oedema improves oxygenation and pulmonary mechanics, V/Q matching largely due to effects on the re-distribution of lung water.

Unilateral Pneumonia and Localized Lung Injuries

High PEEP in these types of patients is more likely to produce overdistension, especially of normally aerated regions because coexistence of areas with normal, low and very low compliance. The net effect of PEEP on gas exchange depends on the balance between over distension of normal aerated alveolar units and recruitment of collapsed (non-aerated) alveoli which is also influenced by the level of PEEP. In patients with unilateral lung disease PEEP may be detrimental whenever it hyperinflates the normal lung, thus directing blood flow to diseased lung producing intrapulmonary shunt.

Development of hypoxaemia depends on total or partial preservation of hypoxic pulmonary vasoconstriction (HPV). In diffused lung injury HPV is partially preserved, which explains lack of hypoxaemia despite of diffuse loss of aeration. HPV may be totally impaired with localized lung injury, which explains severe hypoxaemia despite well preserved lung aeration.

Response to PEEP Titration

The response to PEEP titration varies not only with the type of lung disease but also in relation to gravity.

Type of Lung Disease

- *Diffuse disease*
  When the appropriate level of PEEP is applied to a relatively diffuse disease, alveolar recruitment and distention should improve V/Q without a significant increase in V_D.
- *Non-uniform disease (localized)*
  Application of PEEP to lungs with non-uniform disease can over distend normal alveoli, compressing surrounding capillaries and diminishing perfusion to those alveoli, redirect blood back to the diseased areas [increased dead space (V_D)]. In this situation PEEP can worsen arterial oxygenation.

Gravity Dependent Lung

- *More gravity-dependent lung*
  More gravity dependent lung regions appear to undergo a greater degree of alveolar recruitment, whereas less gravity-dependent lung regions tend to experience greater increases in alveolar volume without recruitment. Radiographical confirmation is available showing that application of PEEP to ARDS affected lungs results in a re-distribution

of tidal ventilation from the less dependent (upper) regions of the lungs to the more dependent (lower) regions of the lungs.

- *Middle lung fields*
  Furthermore, the cyclic alveolar collapses and recruitment that occur with normal tidal ventilation, primarily in the middle lung fields, is reduced with application of PEEP.

CLASSIC ENDPOINTS OF CPAP/PEEP THERAPY

PEEP responsiveness must be determined by the level that provides greatest benefit while producing least non-beneficial effects. There are varieties of method used to determine the best PEEP or optimum PEEP. Best PEEP is the level at which there is an acceptable balance between the desired goals and undesirable side effects. The simplest, safest and most practical method is to use the lowest level of PEEP which maintains the PaO_2 equal to or > 60 mm Hg on a FiO_2 equal to or < 0.6.

Least PEEP

Least PEEP is that which produces an acceptable arterial oxygen tension (PaO_2) or arterial oxyhaemoglobin saturation (SaO_2). This is most frequently used and often adequate.

Maximal Oxygen Transport

Adjusting the PEEP so as to obtain a maximal oxygen transport (arterial oxygen content x cardiac output). This also takes into consideration the PaO_2 and haemoglobin.

Lowest Q_s/Q_t

The PEEP level at which the Q_s/Q_t is lowest without a detrimental drop in cardiac output, the optimal level of PEEP correlates with the lowest level of Q_s/Q_t. Many aim at reducing the shunt fraction to 15% or aim at a PaO_2/FiO_2 ratio of 300 or more. Best PEEP was defined as the PEEP level at which the Q_s/Q_t is lowest without a detrimental drop in cardiac output. This requires use of a pulmonary artery catheter.

Best Compliance (Fig. 33.16)

With the use of PEEP FRC increases and compliances also increases to a certain point.

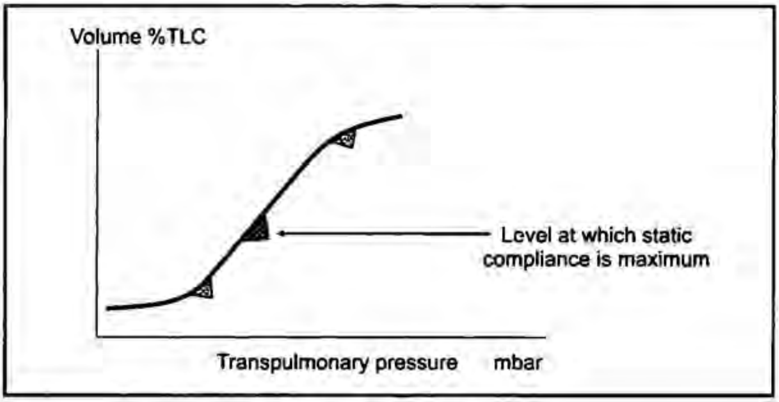


Figure 33.16: Best PEEP

With graded increase in PEEP, there is an increase in static compliance to a certain point and beyond this point it leads to overdistension of alveoli. An optimal compliance produced by particular patients, is generally associated with an optimal rise in the PaO_2.

Lowest V_D/V_T and Lowest $PaCO_2$ – $PETCO_2$

The lowest ratio and the lowest gradient are other parameters for optimal PEEP settings. Best PEEP may also be defined as, that which provides maximum oxygen delivery and the lowest (V_D/V_T) or the level at which static compliance is maximum.

CURRENT RECOMMENDATIONS

1. *Small tidal volume:*
   Current recommendations for mechanical ventilation strategy are based on the use of small tidal volume in order to avoid high-end inspiratory alveolar pressure and alveolar overdistension.
2. *Moderate to high PEEP levels:*
   Recruitment maneuvers may be applied to open closed alveoli. Additionally, use of moderate to high levels PEEP, to keep alveoli open at end expiration, is advised to maintain alveolar recruitment (protective lung strategy).

   PEEP may be applied incrementally to achieve any of the endpoints, or another approach which is based on respiratory physiology, which requires the plotting of the pressure volume curve of the respiratory system.

How to Obtain the Pressure Volume Curve

The simplest technique to obtain the pressure volume curve in a critically ill patient, without having to disconnect the patient from the ventilator, is to inflate the respiratory system by a constant flow (volume-controlled ventilation) delivered by the ventilator; this is a quasistatic technique. The resistive element should be eliminated by using a very low flow rate (< 10 l/min).

Inflection Point

Inflection point is the point of change in the slope of a line. The inflection points represents sudden changes in alveolar opening and closing.

Lower Inflection Point

The lower inflection point represents the opening pressure. Setting PEEP levels at the level of the lower inflection point, is recommended to optimize alveolar recruitment and to prevent repeated opening and closing of alveoli.

Upper Deflection Point

The upper deflection point of the pressure volume curve can be used as a gross estimate of the maximum pressure, which does not cause overdistension in most patients. In those patients with clinical indication of decreased chest wall compliance, higher levels of P_{plat} can be set without increasing the ventilator induced lung injury. However, as recommended, a maximum $P_{plateau}$ should be targeted at about 30 to 35 cm H_2O.

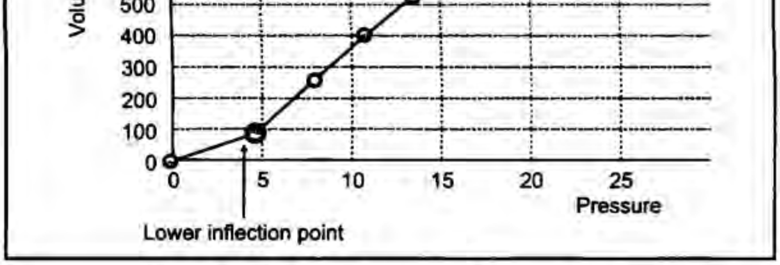


Figure 33.17: Inspiratory volume pressure curve

Use of Inspiratory Volume – Pressure Curve

To Setting Minimum Level of PEEP

Thus, it is clear that inspiratory pressure volume curve of the total respiratory system identifies the minimum level of PEEP (lower inflection point) required for preventing at least partial de-recruitment, although a higher level may be required in many patients.

Setting Tidal Volume or Plateau Airway Pressure

The tidal volume or plateau airway pressure should be set at the upper deflection point. The upper deflection point represents recoiling characteristics. The higher the opening pressure the stiffer the lung, as indicated by the curve moving laterally to the right along the pressure axis. In these patients with clinical indications of decreased respiratory compliance, higher levels of P_{plat} can be set without increasing the probability of ventilator induced lung injury.

CONTRAINDICATIONS OF PEEP

Absolute

- Hypovolemic shock
- Undrained high pressure pneumothorax

Relative

- Unresolved bronchopleural fistula
- Intracranial hypertension and low cerebral compliance
- Chronic chest wall restrictive disorders

APPLICATION OF PEEP OR CPAP IN INFANTS WITH REDUCED COMPLIANCE

1. The application of PEEP or CPAP in infants with reduced compliance receiving mechanical ventilation has to be done with caution, adapted to the pathophysiology and the course of the lung disease, as well as the other lung mechanics.
2. Infants with congenital diaphragmatic hernia and hypoplasia of the lung should be ventilated with—
   - Low inspiratory pressure
   - Short inspiration time
   - Small PEEP of 2-3 cm H_2O

   This will prevent pneumothorax, pneumomediastinum and post-operatively, overdistension of the alveoli and development of air leaks.
3. In children with tracheomalacia PEEP decreases the airway resistance by distending the airways and preventing dynamic compression during expiration.

CHAPTER

34

Pressure Support Ventilation

Pressure support ventilation combines the advantages of pressure controlled ventilation with spontaneous breathing. It is a similar way The way an anaesthetist feels, slow recovery of spontaneous breath of the patient and then manually supports a breath with a ventilated bag, the same way ventilator can support insufficient spontaneous breathing.

PRINCIPLE OF PSV

1. *Work to be done to draw the gas down the tube (Fig. 34.1)*
   A tube having an open proximal end and one manometer at either end. Distal end of the tube is connected to a flow meter, to which a suction regulator aspirating one liter of gas per second is attached. The suction causes atmospheric air to be drawn in, through the open proximal end of the tube.

   The proximal manometer records zero (i.e. atmospheric) pressure while the distal manometer records a negative (i.e. subatmospheric) pressure, due to the drop in pressure along the tubes, in accordance with **Poiseuille's law.** Imagine for a moment that the suction is actually the patient's inspiratory flow and the tube is his airway, WOB is the work that must be done to draw the gas down the tube.
2. *Flow nuilifies the work required to draw gas down the tube (Fig. 34.2)*
   At the proximal end of the tube a flow meter is added, which delivers flow, which is equal to the flow aspirated by the suction. This flow nullifies the work, required to draw gas down the tube. This is analogous to a PSV mode in which the pressure is set, at just that

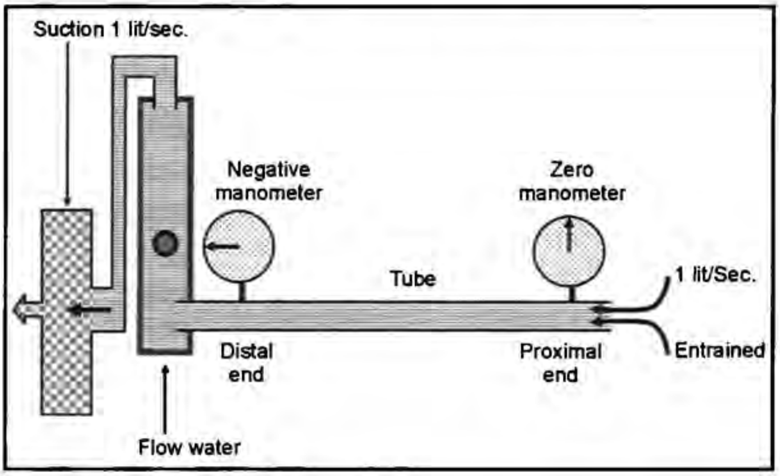


Figure 34.1: Work to be done to draw the gas down the tube (For colour version *see* Plate 16)

level, which would overcome the resistance encountered in drawing gas through the artificial airway and the circuit of the ventilator.

Unlike in the tube and flow meter example the real patient's inspiratory flow rate varies continuously. However, the present day's microprocessors and fast response demand valves are capable of closely

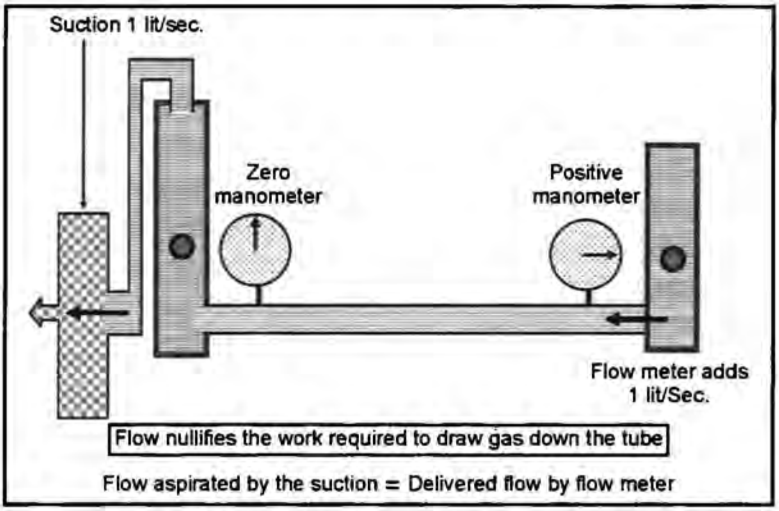

Suction 1 lit/sec.
Zero manometer
Positive manometer
Flow meter adds 1 lit/Sec.
Flow nullifies the work required to draw gas down the tube
Flow aspirated by the suction = Delivered flow by flow meter

Figure 34.2: Flow nullifies the work required to draw gas down the tube (For colour version *see* plate 16)

following changes in the patient inspiratory flow rate and regulating the rate of gas from the ventilator according to the changing needs of the patient.

3. *Flow attenuates elastic workload (Fig. 34.3)*
   The proximal flow meter has been set to deliver a gas flow that is greater than that being drawn down the tube by the suction. This results in the distal manometer registering a positive pressure

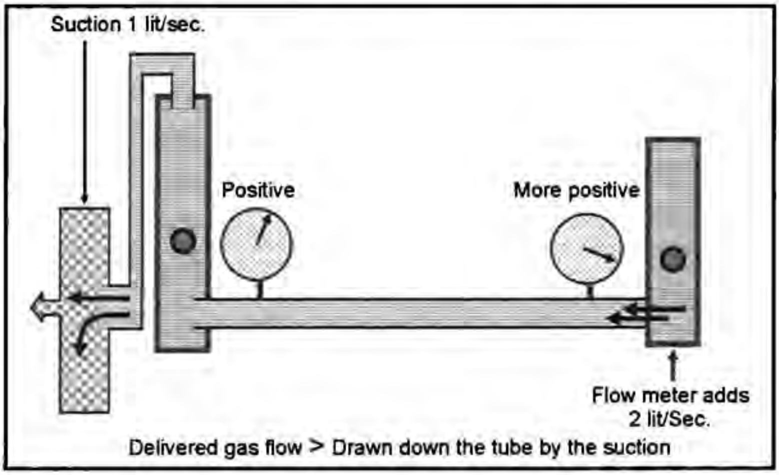

Suction 1 lit/sec.
Positive
More positive
Flow meter adds 2 lit/Sec.
Delivered gas flow > Drawn down the tube by the suction

Figure 34.3: Attenuates elastic workload (For colour version *see* Plate 16)

This is analogous to setting the level of pressure support more than that required to overcome the resistance offered by the artificial airway and the breathing circuit. It is clear that in such a situation, PSV actually assists the inflation of the lung during inspiration, due to attenuation of the elastic workload. Indeed, when the level of PSV is set appropriately high (PSV $_{max}$), WOB can be completely abolished except for the triggering effect.

4. *Normal work of breathing (Fig. 34.4)*

   Work of breathing (WOB), Work = Pressure × Volume

   The crosshatched area represents the elastic workload and the dotted area represents the non-elastic workload. The solid line with the arrowhead is the inhalation loop while the interrupted line is the exhalation loop.

   No work is performed during a normal exhalation (expiration is passive) which is why that loop is "buried" in the area of inspiratory workload.

5. *Pressure support to overcome the resistance (Fig. 34.5)*
   The level of pressure support is just enough to overcome the resistance of the artificial airway and the breathing circuit.

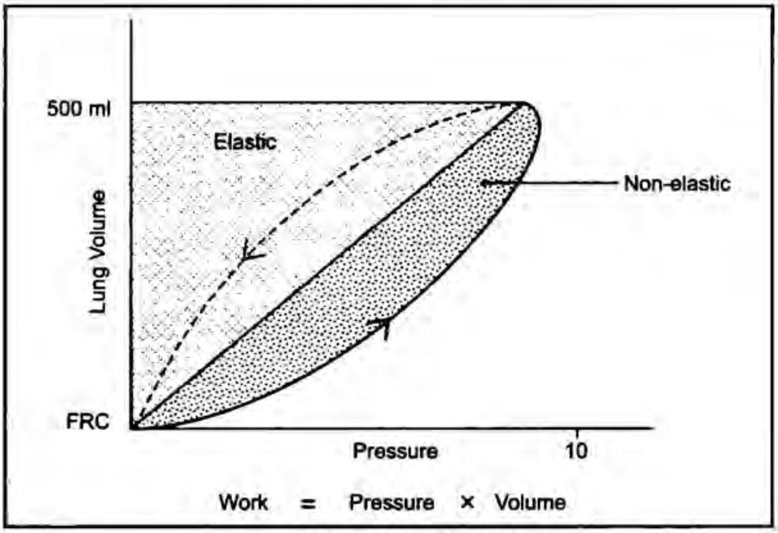


Figure 34.4: Work of breathing

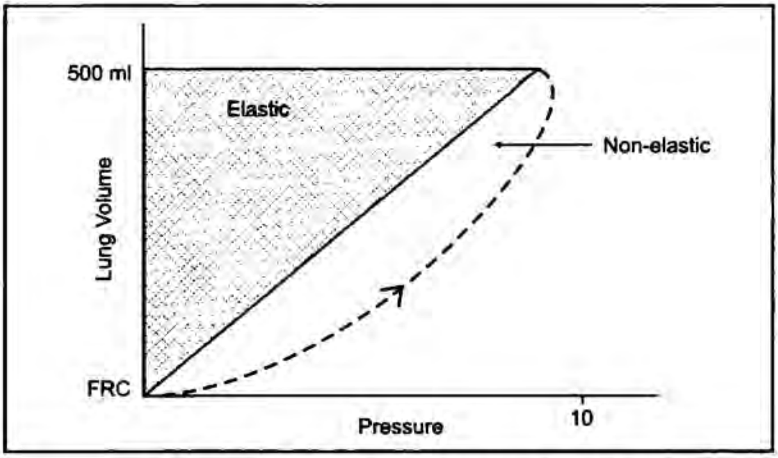


Figure 34.5: Pressure support to overcome the resistance

6. Pressure support has been raised to a level, at which the whole of the non-elastic component and part of the elastic component of WOB have been taken over by the ventilator (Fig. 34.6). Compare with the WOB loops in Figures 34.4 and 34.5.
7. This is PSV_{max}. The entire WOB (except for the work of triggering) has been taken over by the ventilator (Fig. 34.7).

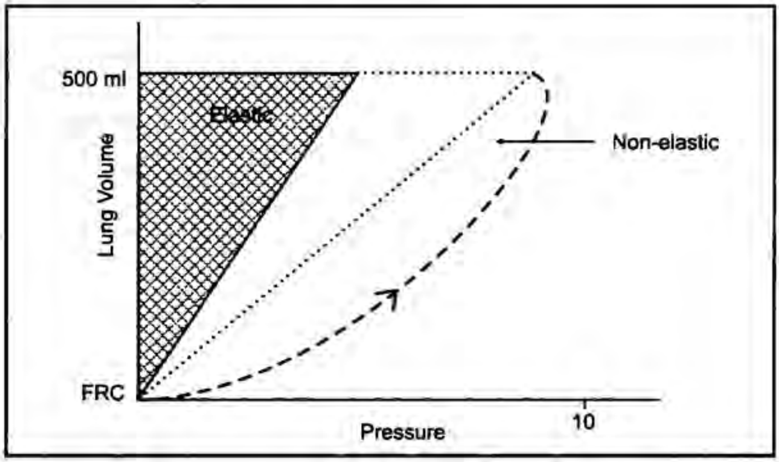


Figure 34.6: Pressure support to overcome the non-elastic and part of elastic components

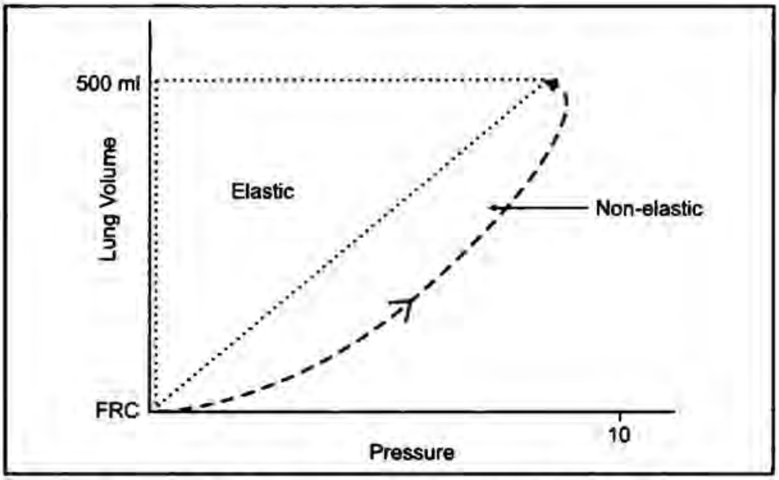


Figure 34.7: Pressure support to overcome entire WOB

INTERACTIVE MODES OR SUPPORT MODES

These modes are termed "interactive or support" modes, because mode permits some degree of spontaneous ventilatory activity, and the patient can affect various aspects of the mechanical ventilator's functions.

- The inspiratory pressure determines ventilation.
- Patient determines respiratory rate, inspiratory and expiratory time and tidal volume.

This method of ventilation is used to augment spontaneous breathing, not to be used as an alternative to controlled or total support modes.

Types of Support Breaths

1. *Pressure support ventilation*-PSV
   Patient triggered, pressure limited with 'flow cycling'
2. *Pressure assist breath*-PAB
   Patient triggered, pressure limited with 'time cycling'
3. *Pressure augmented or volume assured PS breath* – PAPS or VAPS
   Patient triggered, pressure limited, with both flow and tidal volume cycling.

Pressure Support Ventilation (Fig. 34.8)

This mode was first introduced by Norlander in1982 as a part of IMV, but in 1986 it is introduced by MacIntyre as independent mode. Though commonly mode is known as pressure support ventilation, it is also known as:

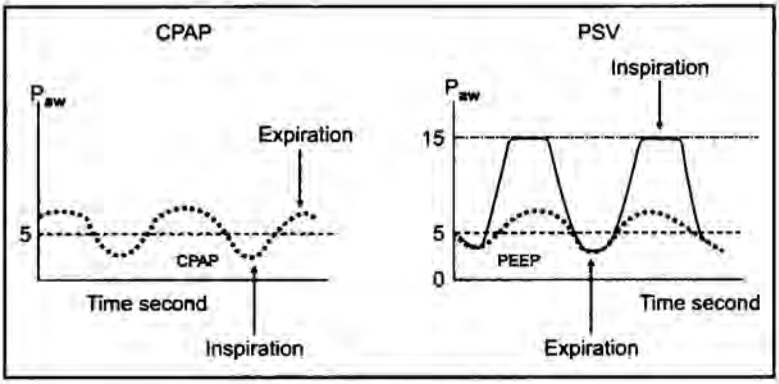


Figure 34.8: CPAP/PEEP

- Assist spontaneous breathing – ASB
- Inspiratory pressure support – IPS
- Spontaneous pressure support – SPS
- Inspiratory flow assist – IFA
- Inspiratory assist – IA

Pressure supported breaths used to be considered as spontaneous because:

- Patient triggered
- Tidal volume varies with the patient's inspiratory flow demand
- Inspiration lasts as long as the patient actively inspires
- Inspiration is terminated when the patient's inspiratory flow demand decreases to a preset minimal value.
- PSV can be used in conjunction with spontaneous breath in any ventilator mode.

However, pressure supported breath is not spontaneous but interactive because: The inspiratory pressure is greater than the baseline pressure.

Parameters to be Set

- Pressure support level above **PEEP**
- PEEP (cmH_2O)
- Oxygen concentration %
- Inspiratory rise time in seconds
- Trigger flow/Trigger pressure
- Inspiratory cycle off % - this is important for the patient's comfort and ventilator synchronization with patient. Inspiratory cycle-off % is the point when inspiration switches to expiration. For a patient with expiratory resistance the inspiratory cycle off should be set to a high value to guarantee enough time for expiration.

Alarms

It is important to monitor the corresponding tidal volume levels with lower and upper alarm limits for expired minute volume.

The maximum time for inspiration in infant is 1.5 sec. and adult 2.5 seconds.

CLASSIFICATION OF PSV

- *Interactive or support mode*
  Patients have considerable control over breath delivery, i.e. flow and timing with pressure support.
- *Trigger*
  Patient triggered, it can be flow or pressure depending on the ventilator.
- *Pressure limited*
  PSV is also pressure limited because the maximum airway pressure cannot exceed the preset pressure support level.
- *Flow cycled*
  It is flow cycled because a pressure supported breath cycles to expiration when the flow reaches a certain level.

COMPONENTS OF A PRESSURE SUPPORT BREATH (Fig. 34.9)

Inspiratory effort being assisted with 20 cmH_2O pressure support (pressure plateau is seen at 25 cmH_2O, since CPAP is of 5 cmH_2O).

The Components of Pressure Support Breath Shows

- Trigger
- Rise time to pressure (pressure support slope)
- Pressure limit (plateau) – Inspiratory pressure

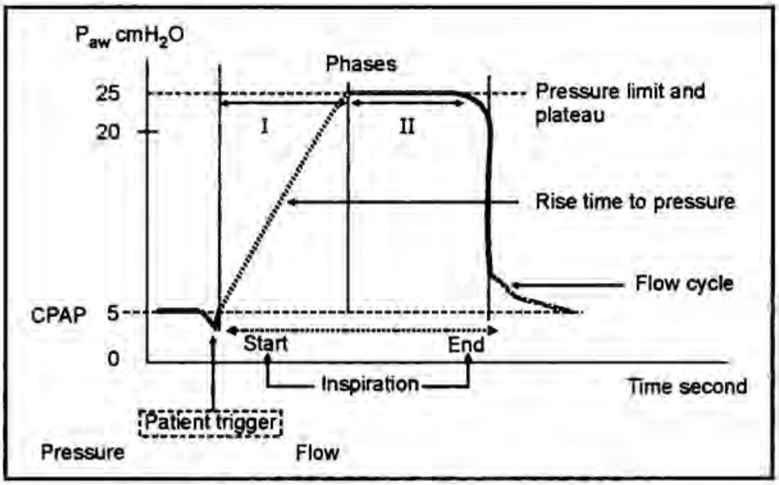


Figure 34.9: Components of pressure support breath

- Cycle variable
- PEEP

Trigger

Triggering of inspiration is initiated by an active effort by the patient and is detected by pressure or flow sensor. Trigger sensitivity is adjustable. Opening of the demand valve can be triggered by a fall in pressure or difference in the flow signal between inspiratory or expiratory flows (referred as flow-by). The opening time delay varies between 50 and 250 ms.

Flow Trigger

For flow trigger, constant flow is delivered to the circuit, during even the expiratory phase. The inspiratory effort is then detected as a small difference between inspiratory and expiratory flows.

Pressure Trigger

The patient's inspiratory effort creates a subatmospheric (negative) pressure in the ventilator tubing. This negative pressure is detected by a pressure sensor that activates a demand valve.

Inspiratory Rise Time (Fig. 34.10)

Inspiratory rise time is a time taken (speed) to reach set peak inspiratory flow or pressure at the start of each breath.

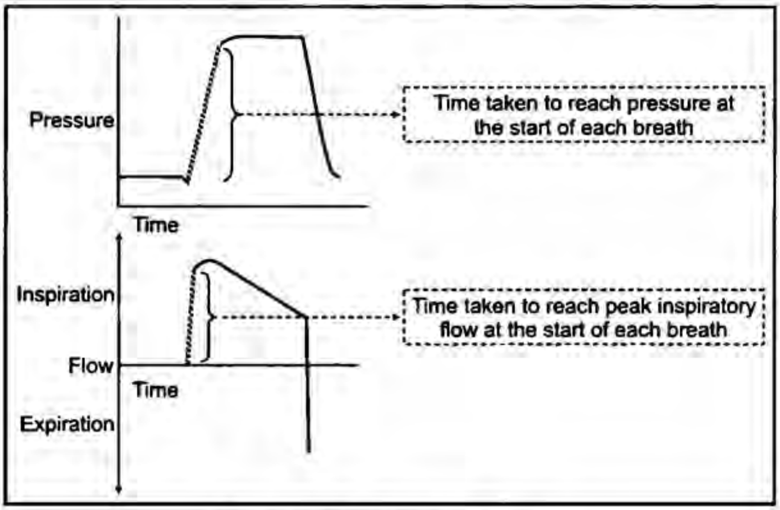
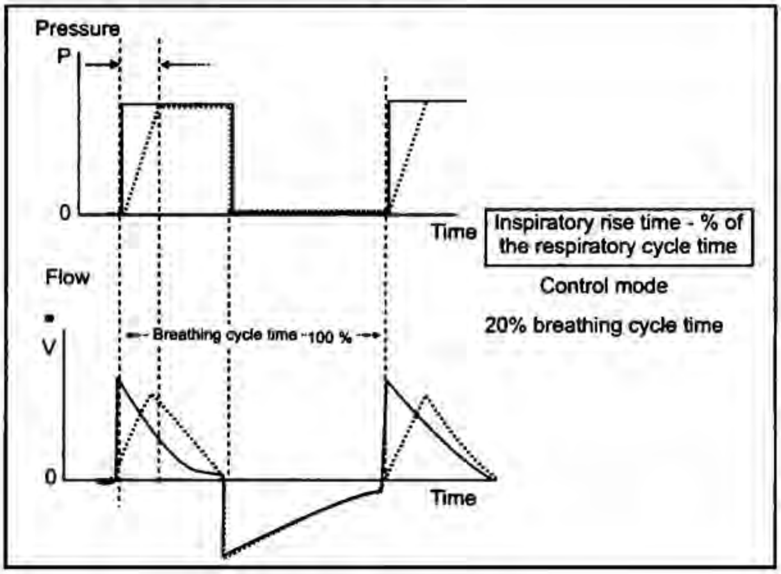

Pressure
Time
Time taken to reach pressure at the start of each breath
Inspiration
Flow
Time
Expiration
Time taken to reach peak inspiratory flow at the start of each breath

Figure 34.10: Inspiratory rise time

In many ventilators, this is preset and nonadjustable. Inspiratory rise time can be as a % of the respiratory cycle time or in seconds or nonadjustable.

- *% Respiratory Cycle Time (Fig. 34.11)*



Inspiratory rise time - % of the respiratory cycle time

Control mode

20% breathing cycle time

Figure 34.11: % Respiratory cycle time in pressure control breath

% Respiratory cycle time is applicable in *control modes* such as, pressure control, volume control, PRVC, SIMV volume control, SIMV pressure control, and SIMV PRVC. Setting can be in the range 0-20% of the respiratory cycle time.

Inspiratory Rise Time in Seconds (Fig. 34.12)

Inspiratory rise time in seconds is applicable in *support modes* such as pressure support, volume support and Bi-vent. For adults the range is 0-0.4 seconds and for infants the range is 0-0.2 seconds. Increase in a rise time can be evaluated by the shape of the flow and pressure waveforms.

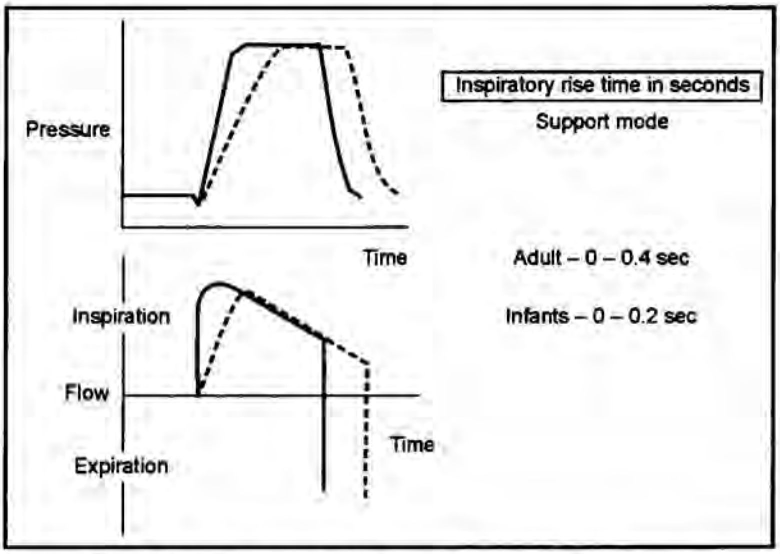


Figure 34.12: Inspiratory rise time in pressure support breath

As rise time increases, valve responsiveness increases and the rise time to the pressure limit is decreased. It is important to set the inspiratory rise time to a comfortable value for the patient. Patients with high ventilatory drives may synchronize better with faster rise times. Patients with less ventilatory drives may synchronize better with slower rise times.

Too Fast Speed

If the pressure increase is too fast, it leads to a very high initial flow. The result is that the breathing cycle is interrupted too early because the criteria (less than 25% of maximum flow) is reached too early, i.e. too fast speed "overshoot" of the pressure limit can occur and premature cycling may result.

Too Slow Speed

If the chosen rate of pressure increase is too small, the given pressure level is reached late. The patient has to do more breathing effort and the patient's "work of breathing increases".

Preselected Inspiratory Pressure – (Fig. 34.13)

The pressure level can be adjusted between 0 (spontaneous breathing through ventilator circuit) safely upto maximum of 30 cmH_2O, after the start of spontaneous ventilation. The absolute pressure support is therefore calculated from the difference between reference pressure i.e. pressure support and PEEP. However, in some ventilators the pressure support is already given in cmH_2O above PEEP level. These instrument specific differences have to be borne in mind in the application of pressure support.

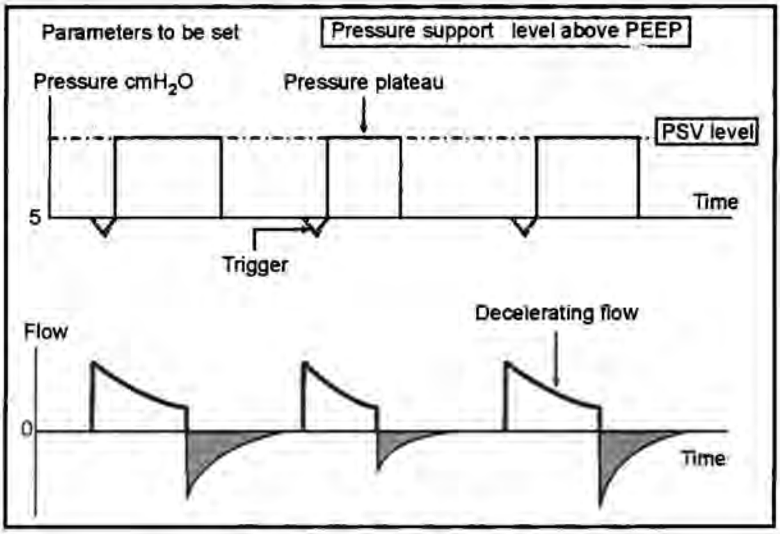


Figure 34.13: Pressure limit - Plateau

Preset or Constant Pressure

Once triggered (flow or pressure), the ventilator demand valve generates flow high enough to increase the airway pressure to the preset pressure limit, expiratory valve remains closed until the preselected airway pressure is attained. The preset (constant) pressure maintains the pressure plateau for the duration of the patient's spontaneous effort.

Decelerating Flow

Inspiratory flow decreases rapidly throughout the rest of inspiration (demand valve regulates the flow continuously via microprocessor servo

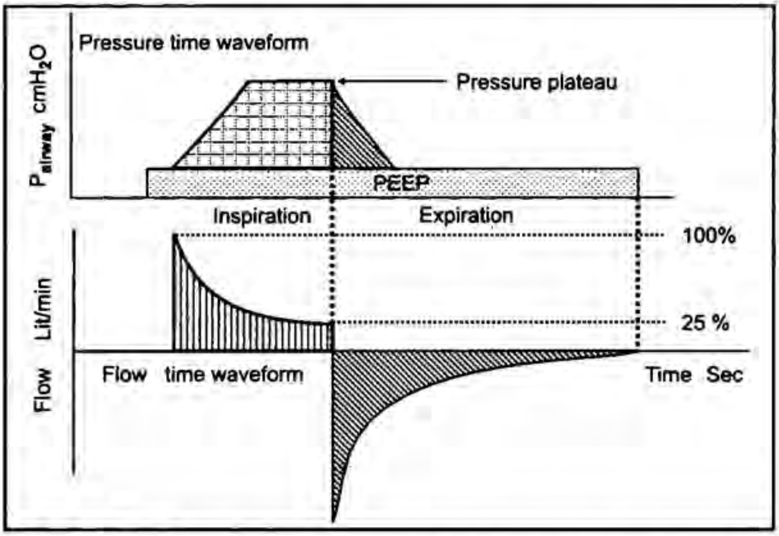


Figure 34.14: Pressure/Flow time waveform in pressure support breath

control) to keep this pressure constant until expiration occurs. Alternatively, inspiratory flow ceases at a specific flow rate.

Cycling to Expiration

During PSV, cycling to exhalation is triggered primarily by a decrease in inspiratory flow from its peak to a system specific threshold value. This critical decrease in inspiratory flow is taken as indirect evidence that the inspiratory muscles have started to relax.

Types of Cycle Variable

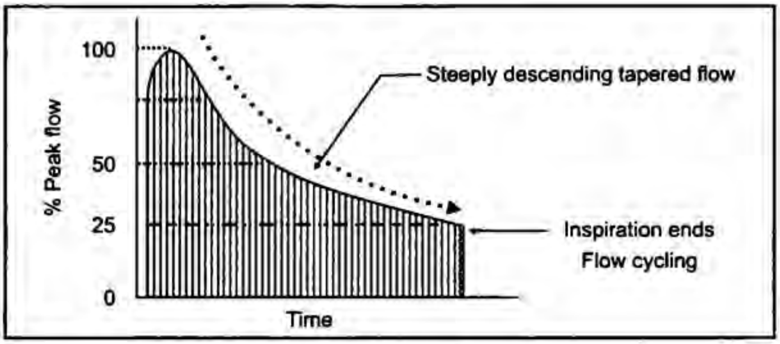
Flow Cycling (Fig. 34.15)

Inspiration is terminated when peak inspiratory flow rate reaches a minimum specified level.

Expiration is triggered either 25 % of initial peak inspiratory flow or an absolute level of flow (between 2 and 6 lit/min) depending on ventilator settings. However some ventilator allows the flow cycle variable to be set at a percentage of the initial peak flow, adjustable from 10% to 40%.

Extra Safety Mechanism

However, in the interest of patient safety, other cycle variable are included such as "pressure and time".



Flow 34.15: Flow cycle variable

Pressure cycling

Flow through the valve being terminated when the airway pressure exceeds the level of predetermined pressure alarm setting. Detection of a small degree of pressure (1-3 cm H_20) above the fixed pressure support level, consequent to sudden expiratory effort by the patient, also can be used (alone or in combination with flow criteria) to terminate inspiratory assistance. This backup protects the patient's lung from barotrauma if he coughs or strains.

Time cycling

Time-cycling backup prevents a prolonged inspiration in the event of significant leak in the breathing circuit of the ventilator. Time limit for inspiration serves as a safety mechanism if a leak develops in the circuit and two previous methods of terminating inspiration become inoperative. Complications have been reported in the absence of this time limit mechanism, whereby constant insufflations (at the PS level) creates high level of continuous positive airway pressure (CPAP).

During a pressure support breath, the longest allowed inspiratory time is usually 3 seconds. This prevents prolonged inspiratory time when a low flow cycle criteria is used (i.e. 5 L/min) in the presence of a air leak. In most ventilators, these cycle variables are buried in the software.

PHYSIOLOGICAL EFFECTS OF PSV

1. *Breathing pattern*

   Addition of pressure support seems to allow the patient to breath in a physiological and natural way but also modifies the spontaneous breathing pattern. The higher the preset inspiratory pressure level, the more gas flows to the patient. Most patients develop an increase in V_T and decrease in respiratory rate with increasing levels of pressure support. There is an inverse relation between the PS level and respiratory rate and a direct relationship with tidal volume. As the patient becomes more active the pressure support level may be gradually reduced.

Clinical Importance of Change in Breathing Pattern

Pressure support allows the patient to maintain full control over the respiratory rate and partial control on inspiratory time and tidal volume. This suggests that the breathing pattern adapts rapidly when the workload of respiratory muscles changes. It signifies that adjustment of the PS level can be guided by observing the breathing pattern response, which usually occurs within 1-2 minutes.

Excessive level of pressure support, however, leads to hyperinflation, respiratory alkalosis, respiratory depression and a period of apnea. High level of support induced hyperinflation may result in an inability to trigger the ventilator (so called ineffective triggering) giving rise to fatigue.

2. *Gas exchange and distribution of ventilation and perfusion*

   The primary goal of PS is to support the patient's effort while delivering a satisfactory gas mixture. PS is not aimed primarily at improving oxygenation. The effects of PS on gas exchange are explained primarily by increased alveolar ventilation resulting from changes in the breathing pattern. Other factors may influence arterial blood gases, due to changes in oxygen consumption, modification of total dead space and altered distribution of ventilation due to PS.

3. *Work of breathing and respiratory effort*

   A major goal of PS is to assist respiratory muscle activity in a way that improves the efficacy of the patient's effort and to decrease workload. The level of PS can be titrated so that the respiratory muscles can offload a part or the whole of WOB.

   During breathing activities, pressure varies continuously in magnitude and direction, while at the same time volume varies continuously as a function of pressure. There are three essential components in this opposition:

   - Elastic resistance
   - Air resistance
   - Structural resistance

   Increased airway resistance and decreased chest wall or lung compliance increases the transpulmonary pressure (P_{tp}) required to inflate the lung. Expiration is normally passive, but in patients with lung diseases it may become active. The expiratory work must then be included in total WOB.

The neuromuscular drive to breathe is high during the acute phase of respiratory failure and remains high until the patient can be weaned. The bio-engineering response to increased impedance is to delay the onset of fatigue by minimizing the required increase in driving pressure. This is achieved by shorter T_I, thus reducing V_T and increasing frequency, since the WOB is proportional to V_T and f.

WOB refers to the work generated by the patient or ventilator to overcome the elastic and resistive properties of the respiratory system.

Work (WOB per unit time) = P cm H_2O × MV (minute volume in ml)

P - is the total pressure change during the respiratory cycle

- System is patient-cycled, therefore the duration of inspiration and expiration and total cycle (T_I, T_E, and T_{TOT}) are determined by the patient.
- Therefore, the mean inspiratory flow rate ($= V_T/T_I$) is also partially determined by the patient.
- This is a measure consideration because power (WOB per unit time) equals pressure applied (P) × minute volume.

$$
\begin{aligned}
WOB &= P \times MV \\
&= P \times \text{Tidal volume} \times RR \\
&= P \times (\text{Flow rate} \times T_I) \times f \\
&= P \times [(V_T/T_I) \times T_I] \times 60/T_{TOT} \\
&= P \times [(V_T/T_I) \times (T_I / T_{TOT}) \times 6o]
\end{aligned}
$$

Where $[(V_T/T_I) \times (T_I / T_{TOT}) \times 6o]$ is, in effect, minute volume

RATIONALE OF USING PSV

1. The rationale is that PS will prevent fatiguing by reducing the patient's contribution to P while maintaining an appropriate V_T/T_I, T_I and T_I/T_{TOT}
2. It has been shown that patients who cannot be weaned have the same V_T/T_I as the ones who can, but that T_I is less in the former, leading to shallower and faster breathing.
3. PS provides breath to breath support or assistance by means of 'positive pressure boost' synchronized with inspiratory effort. The pressure-augmented breathing allows the patient to determine the inflation volume and respiratory cycle duration.

GOALS OF SETTING ADEQUATE PRESSURE

The total work reflects, work required to overcome the impedance (resistance and compliance) due to disease process and also work of breathing imposed by the artificial airway and the ventilatory circuitry.

- The setting of pressure support should be that required to overcome the imposed increased impedance plus to increase the tidal volume (V_T) if necessary.
- Thus for the same patient effort, minute ventilation (V_E) will increase at the same or even at a reduced frequency (f). Reduction of the respiratory rates accompanied by reduced respiratory efforts resulting in more efficient breathing.

DIFFERENT INTERACTIONS OF PRESSURE SUPPORT ON RESPIRATORY SYSTEM

Effect of pressure support to spontaneous breath has several effects on the respiratory system that are different from unsupported spontaneous or other types of ventilator delivered breaths. These differences involve two broad areas:

- PS interaction with ventilator muscle function
- PS interactions with patient flow demand (synchrony)

Ventilatory Muscle Function

Pressure support; by providing inspiratory boost add to the patient's own efforts to deliver the V_T (Fig. 34.16).

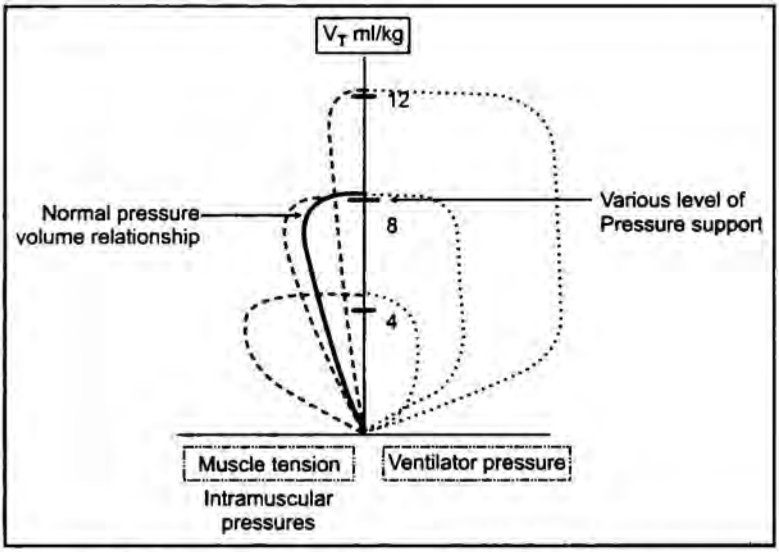


Figure 34.16: Patient's and ventilator's contributions of the work of breathing during various levels of pressure support

The efforts of various levels of applied pressure support are represented by solid lines. Patient work is progressively shifted to the ventilator as the level of pressure applied with every breath increases. The pressure-volume work characteristics are also changed with increasing pressure support to a normal configuration.

The patient's response to 'pressure boost' can be different from the patient to patient and depends on ventilator muscle strength and endurance capabilities. A severely overloaded ventilatory muscle is usually characterized by tachypnoea and small tidal volumes. Adding a small amount of PS to such a patient tends to augment each effort the patient makes, to increase tidal volume and decrease in respiratory rate with increasing levels of PS.

Shifting Work from Patient to Ventilator

Overall the patient work is reduced by decreasing the required breath rate. As higher level of PS tidal volume approaches around 4-7 ml/kg range, the patient's contribution to each breath begins to decrease and tidal volume tends to remain constant. Ventilatory muscles work during this range of PS is thus reduced because tidal breath work is being shifted from patient to ventilator.

Shifting of Work to Lower Value

In addition the patient work is shifted from patient to ventilator; work is also shifted to a lower value which is more favorable from an energy point of view. Once enough PS has been given to reduce patient work per breath to low levels (usually associated with tidal volume 7-9 ml/kg), additional PS only serves passively to increase in the tidal volume. From this it is clear that different amount of PS behave differently in different patients, depending on the state of ventilatory muscle. As the patient becomes more active the pressure support level may be gradually reduced. For example 20 cmH_2O may be just enough to relieve fatigue in a patient with poor compliance whereas it overdistends the lung of patient with normal lungs. PS setting should be adjusted to response (Rate/V_T) rather than arbitrary value.

Patient Ventilatory Synchrony

The ventilator's sensitivity and responsiveness can contribute to the dyssynchrony. Because PS is interactive breath, one must consider the three mechanical breath phases (trigger, limit, and cycling) and how their synchrony is achieved with patient effort.

Trigger Synchrony

Trigger synchrony means assuring prompt support breath to initiation of the patient's demand. A certain amount of dys-synchrony exists on all mechanical ventilators due to separation of the ventilator muscles and ventilator's sensor and flow controller. Trigger responses have improved in new ventilators with the use of slope adjustors or rise time adjustors. An imposed triggering load can result from intrinsic positive end-expiratory pressure developing in patients with air flow obstruction.

Flow Synchrony

Flow synchrony means ventilator gas delivery in accordance with patient efforts. It should be continuous and adequate throughout the breath. Flow dys-synchrony is the result of inadequately delivered inspiratory flow. This is a particular problem with conventional volume cycled breath that have fixed flow pattern. This is more significance especially in patients being weaned with synchronized mandatory ventilation (SIMV). An important feature of a pressure limited breath is a rapid and variable flow that tends to match the patient's demand better. This may be further enhanced by the pressure slope or rise time adjustment.

Cycling Synchrony

Cycling synchrony refers to matching ventilator breath termination with the end of patient effort. Normally flow cycle in a PS breathe at 25-35% of peak flow which is reasonably normal in most patients. But both premature and delayed termination can produce cycling disharmony.

- *Premature or early termination:* Early termination may result if the:
  - PS level is too low
  - The initial flow is particularly fast (such that the 25-35% flow cutoff occurs quite early in the breath)
  - A patient expiratory reflex develops quickly in response to pressure. Clinician adjustment of the pressure level or the inspiratory rise time may be helpful.
- *Late termination:* Late termination can produce a different type of dys-synchrony which is usually a result of an excessive PS setting but it may also occur if the initial flow delivery is too low (thus late occurrence of the cycling criteria). In addition to patient discomfort, an important consequence of delayed termination is inadequate expiratory time and intrinsic PEEP and air trapping. Proper assessment of PS level and inspiratory rise time usually alleviates this problem.

Principle for Conditioning of Respiratory Muscle

Work of breathing during partial ventilatory support is a major determinant deciding weaning success or failure. The success or failure of discontinuation of ventilatory support (weaning) depends on rehabilitation and endurance training of the muscle of respiration. It has been claimed that PS helps the respiratory muscles to be conditioned for endurance and strength, during gradual discontinuance of ventilatory support.

Endurance Conditioning

A respiratory muscle needed during weaning is endurance conditioning which is not unlike running a marathon over several hours. It is achieved by continuous repetitive low-level work.

Strength Conditioning

Strength conditioning involves maximal work for short periods with complete rest in between; it is somewhat like sprinting over a short distance. The T-piece technique of weaning from mechanical ventilation that was popular till 80's provides this kind of conditioning.

CURRENT APPROACHES TO THE CLINICAL APPLICATIONS

The three application of PS currently being used are:

Low Level of PS

The airflow resistance associated with an endotracheal tube produces an undesirable workload that may compromise comfort of the patient and ventilatory muscle function during spontaneous breaths. Low level of PS, i.e. 2-10 cmH_2O pressure can be used during inspiration to overcome the resistive component of inspiratory work, imposed by an endotracheal tube. Thus, this use of PS may be indicated in any intubated patient, who is taking spontaneous breaths and in which tachypnoea; dyspnea or ventilator patient asynchrony is felt at least partially due to the spontaneous ventilatory muscle work imposed by the endotracheal tube.

Low levels of PS 7-10 cmH_2O is as efficient as a T-piece trial.

Weaning Protocol Using PS

In patients requiring prolonged ventilatory support pressure support provides more comfortable unloading of ventilatory muscle and more physiologic work, than intermittent approaches using spontaneous breath such as T-piece technique. This mode is more useful in those patients who are difficult to make comfortable and who will require a prolonged weaning process because of slowly resolving lung disease.

Higher Level of PS in Early Part of Weaning

The initial pressure support level should be chosen in such a way, that it unloads the respiratory muscle completely. Higher level of PS can provide substantial ventilatory support by applying the necessary level of inspiratory pressure for a desired tidal volume and minute ventilation. A useful initial setting for PS under these circumstances is one that provides whatever inspiratory pressure is necessary for V_T of 7-9 ml/kg. This level of PS can reduce the patient's work to nearly zero.

Reducing PS Level Guided by Respiratory Rate

With the resolution of lung disease, the pressure support level can be gradually decreased allowing the patient's respiratory muscles to increase the level of work of breathing. Weaning of PS from this point is accomplished by reducing the level of PS, which can generally be guided by the patient's respiratory rate (an indicator of the patient's own sense of load). Mechanical ventilation is discontinued when the pressure support level is about 5-6 cmH_2O.

Elevation in respiratory rate often indicates an excessive ventilatory muscle load and thus represents more ventilatory support in the long term weaning of a patient. Increase in night support to facilitate sleep may be beneficial.

Non-invasive Application of PS

Most of the systems for non-invasive ventilation can deliver up to 20 cm H_2O of PS or more. PS can be used via face mask systems in non-intubated patients who require transient ventilatory support (overdose, asthma, postoperative, exacerbation of chronic obstructive pulmonary disease).

CLINICAL USES

PSV must be used only in those patients who have an intact respiratory drive.

1. *Stand-alone mode*

   Pressure support can be used as a stand-alone mode, only those with a reliable ventilatory drive and stable ventilatory requirements. In this every breath is patient triggered and close monitoring is required. A patient with unstable ventilator drive or rapidly changing lung impedance is prone for suboptimal alveolar ventilation.

2. *To predict patient tolerance of unassisted breathing and extubation*
   The idea is based on selecting a level of PS which is just sufficient to overcome the circuit resistance. Thus spontaneous muscular activity should be similar to what a patient would perform in the absence of an ETT or circuit. Measurement of breathing pattern is of major help in assessing work of breathing or ability to tolerate extubation. Therefore, the pressure needed to obtain a reasonable breathing pattern can provide insight into a patient ability to tolerate extubation.
   The work of breathing to overcome the tracheal tube resistance and demand valve inertia can be quite considerable. Pressure support required to eliminate the additional work of breathing is shown:-

ETT (diameter mm)	7.5	8	9
PSV level (cmH_2O)	8.0	6.0	4.0

3. *PSV with SIMV*
   It significantly lowers the oxygen consumption requirement presumably due to the reduced work, and augments a patient spontaneous tidal volume.

4. To lower the work of spontaneous breathing to facilitate weaning in a difficult to wean patient
   PS with PEEP can be used in patient who do not have enough muscle power (pulmonary pump weakness), e.g. for weaning from the ventilator support.
   In patients with underlying lung disease, the PS level that compensated for the additional work ranged from 8-14 cm H_2O, whereas it averaged 5-10 cmH_2O in patients of free lung disease.

5. *Non-invasive ventilation – in COPD patient*
   PSV can be used in specialized facemasks or nasal masks with inflation pressures of 20 cmH_2O. PSV decreases respiratory muscle activity markedly in patients with COPD in acute distress. The intrinsic PEEP which occurs with the tachypnoeic COPD patient can be effectively reduced with PS. This is primarily due to the fact that PS reduces the breathing frequency. This results in an extension of breathing cycle, and therefore of the expiration time, as well as reductions of oxygen demand. The shortening of the inspiratory time (expiratory valve is opened by the flow control as soon as the flow falls below 25% of the maximum flow) especially benefits COPD patients. When the patient's own respiratory effort is increasing again, i.e. when ventilator dependence reduces, the pressure assistance can be reduced by gradually reducing the inspiratory level.

During exercise, which also constitutes a high ventilatory workload an invasive PS improves performance in patients with COPD. During the weaning of patients with hypercapnic respiratory failure, addition of PS produced correction of $PaCO_2$ and respiratory acidosis.

6. *In infants and children*

   In infants and children an endotracheal tube and a demand valve in the ventilator circuit are additional loads in the WOB. In small Paediatric patients the inspiratory work required to decrease the pressure in the ventilator circuit and open the demand valve is not changed even when adding pressure support.

   PSV seems to a good adjunct in the older infant, weighing more than 3-4 kg.

   Usually use PS 5 cmH_2O for older patients and 10 cmH_2O for smaller patient (smaller ETT has higher resistance, more impediments to flow.

TITRATION OF THE PROPER LEVEL OF PRESSURE SUPPORT

Desired Tidal Volume

Pressure support level can be increased until a desired spontaneous tidal volume is achieved (e.g. 8 to 10 ml/kg).

To Secrease Spontaneous Respiratory Rate

Pressure support level can be increased until the patient's spontaneous respiratory rate decreases to a target value (usually 25 breaths/min or less).

To Decrease the Work of Breathing

This approach is probably less commonly used for the patient in immediate respiratory distress but is more often used as a "routine" method to decrease the WOB. Since an ETT provides an increased airway resistance and increased WOB, pressure support has been successfully used to overcome this gas flow resistance.

PRECAUTIONS FOR SELECTING PSV

1. Pressure support level of less than PSV_{max} may result in
   - Diaphragmatic fatigue
   - Use of accessory muscles
   - And failure to wean
2. Pressure support level of greater than PSV_{max} may cause
   - Hyperinflation of the lungs
   - A decrease in the venous return (with its consequences)

PATIENT MANAGEMENT

Once pressure support is established

- The level of pressure support should be reduced at a speed tolerated by the patient. The institution of pressure support frequently leads to a significant reduction in the respiratory rate and in the use of the accessory muscles of respiration.
- If airway reflexes are intact, the patient can be extubated at a pressure support level of 5-7 cmH_2O because that is the level required to overcome the resistance offered by artificial airway and the ventilator circuitry.

CONTRAINDICATION OF PSV

- In patients who need sedation and muscle relaxation
- In patient with central neurologic disorders
- In patients without ventilatory drive, e.g. hypoventilation syndrome

ADVANTAGES OF PSV

- Increase in the tidal volume and reduction in the respiratory rate
- Reduced work of breathing—reduced oxygen consumption
- Reduced respiratory muscle loading, resulting in more efficient breathing
- No need for sedation
- Good ventilatory comfort
- Better patient ventilator interaction

CONCLUSION

PSV is a mode of partial ventilator assistance that has proved very efficient in reducing the work of breathing and providing relatively good synchrony with patient effort. It can be also used early in the course of ventilation, enabling the patient to be ventilated without the need for sedation and in preparation for weaning. Its main drawbacks, however, is the possibility of overassisting patients causing major dys-synchrony between patient rhythm and that of the ventilator inducing hyperventilation. Studies comparing SIMV and pressure support ventilation aiming to achieve similar tidal volumes reported lower peak airway pressures, lower mean airway pressures and lower work of breathing with PSV mode.

CHAPTER

35

Volume Support Ventilation, Pressure Regulated Volume Control Ventilation, Automode

VOLUME SUPPORT VENTILATION

Volume support breaths are *'Volume assured pressure support' VAPS.* It combines volume targeted ventilation with pressure support in spontaneously breathing patients. This is another modification of pressure support ventilation where servo control maintains a target tidal volume.

SPECIAL FEATURES

Essentially mode is like PRVC, but available in spontaneously breathing patients. Like PRVC one can set a desired tidal volume in the volume support mode and the level of pressure support is continuously adjusted by the ventilator.

The patient has control of the inspiratory phase as in pressure support, where as with PRVC, inspiratory time, I:E ratio and rate are to be set. As in the pressure support mode, each breath is patient triggered, pressure limited, and flow cycled and flow is decelerating.

The volume support mode is where the patient will be given support in proportion to his inspiratory effort and the target tidal volume. If the patient's inspiratory activity increases the inspiratory pressure support will decrease, to maintain the set tidal volume. If the patient breathes below the set tidal volume, the inspiratory pressure support will increase.

The pressure support level varies at each breath to achieve the target tidal volume.

VENTILATORY PARAMETERS

- ***Tidal volume***
- ***Set upper pressure limit***
- PEEP
- Trigger flow/pressure
- Inspiratory cycle-off %

Similar to PRVC, it performs initial test breaths (low pressure) to calculate compliance and then administers additional test breaths at a higher pressure before going to the calculated pressure. The inspiratory pressure is controlled automatically between PEEP and set upper pressure limit (Fig. 35.1).

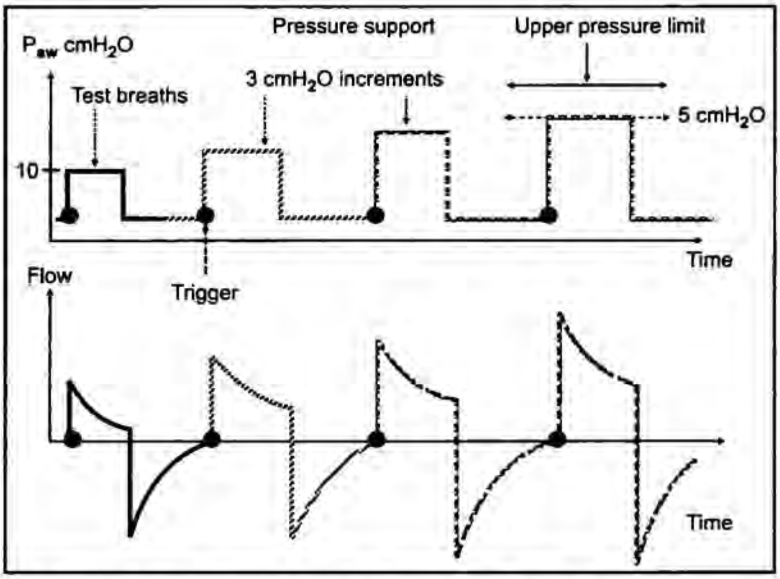


Figure 35.1: Test breath with low pressure followed by additional test breaths with higher pressure

The start breath is given with pressure support of 10 cmH_2O. From that breath the ventilator calculates and continuously regulates the pressure needed to deliver the preset tidal volume. After the first breath the pressure increases or decreases in steps of maximum 3 cmH_2O. During the remaining 3 breaths of the start up sequence the maximum pressure which will increase, is upto 20 cmH_2O for each breath. If the delivered tidal volume decreases below the set tidal volume, the pressure support level is increased in steps of maximum 3 cmH_2O until preset tidal volume is delivered. If pressure support level causes larger tidal volume than preset, the support pressure is lowered in steps of maximum 3 cmH_2O until the preset tidal volume is delivered (Fig. 35.2).

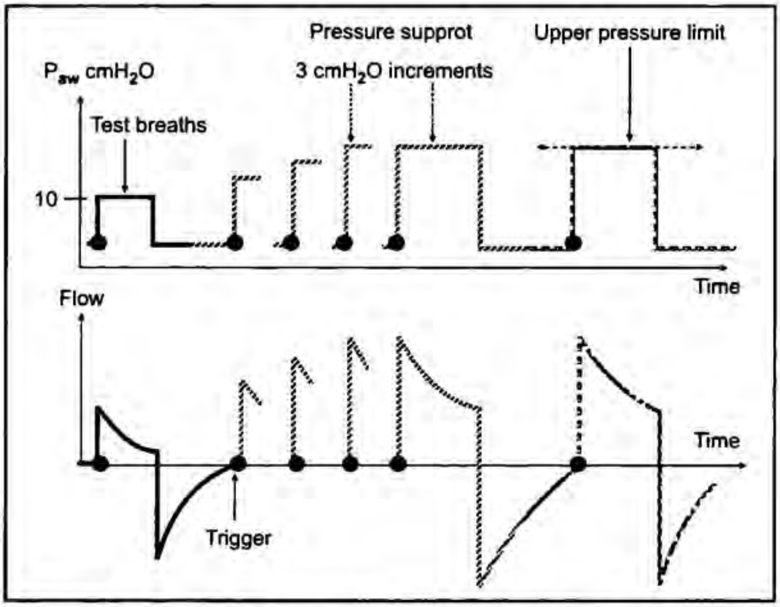


Figure 35.2: Increment of pressure support to achieve targeted tidal volume

INSPIRATION STARTS

- Inspiration with volume support starts when the patient triggers.
- Volume support assures a set target tidal volume upon patient effort, by an adapted inspiratory pressure support.
- The inspiratory pressure level is constant during each breath, but alters in small increments, breath by breath, to match the patient breathing ability and lung mechanical properties.

EXPIRATION STARTS

- When the inspiratory flow decreases below preset fraction of the inspiratory peak flow (flow cycle).
- If the upper pressure limit is exceeded.
- Maximum time for inspiration is exceeded.
  The maximum time for inspiration is: infant 1.5 sec. and adult 2.5 sec.

ALARMS FOR VOLUME SUPPORT

- *Minute volume alarm*
  In all spontaneous modes it is important to set the minute volume alarm.
- *Apnoea alarm*
  In this mode it is also important to set the apnea alarm depending on the patient. If apnoea duration is reached or if spontaneous RR

falls below apneic alarm levels then the ventilator will automatically switch to *'Back up mode'* providing controlled ventilation, i.e. automatically switches to PRVC.

ADVANTAGES

This mode gives the lowest inspiratory pressure level necessary to deliver preset volume to patient's breathing spontaneously.

PRESSURE REGULATED VOLUME CONTROL VENTILATION

Pressure regulated volume control ventilation (PRVC) is a dual control mode, which is modification of pressure control ventilation, which can be controlled or assisted. This is a form of closed loop ventilation that has the combined feature of volume and pressure controlled ventilation. PRVC has been compared to your being there breath by breath, hour by hour, day and night, ventilating the patient with your "educated Hand" regulating the inspiratory pressure and controlling the tidal volume.

CLASSIFICATION

PRVC is pressure controlled mode, however it has characteristics of a volume controlled, assist-control mode. As in the pressure control mode, each breath is mandatory or pressure triggered, pressure limited and time cycled. PRVC adjusts the flow rate for the set tidal volume at or below the set maximum pressure.

SPECIAL FEATURES

In PCV one has no control over tidal volume and the only way to increase the tidal volume is by increasing inspiratory pressure.

PRVC has both the benefits of pressure control ventilation, and volume control ventilation (a constant tidal volume), and automatic weaning as patient compliance improves and/or patient effort increases.

In PRVC, one can set a desired tidal volume and the level of pressure control is continuously reset by the ventilator using data from the last few breaths to deliver the target tidal volume.

VENTILATOR PARAMETER

- ***Target tidal volume (ml) or Minute volume (lit/min).***
  The ventilator attempts to achieve the volume target using a pressure control gas delivery at the lowest possible airway pressure.

- *Maximum inspiratory pressure.*
- *I:E ratio/Inspiratory time.*
- Respiratory frequency.
- PEEP.
- Trigger flow/Trigger pressure.

FUNCTIONING OF PRVC

Test Breath – Volume Controlled Breath

Starting of the mode the first delivered breath is a test breath (volume controlled breath), at some minimal pressure level (5-10 cmH_2O), which is used to calculate patient compliance.

Next Few Breaths – Pressure Controlled Breath

- The next few breaths may be delivered at a pressure, below the calculated pressure needed to deliver the target tidal volume, as a further test breath and after this calculated pressure is applied.
- If the target volume is exceeded, the pressure limit is decreased by 1-3 cm H_2O on each breath until the target volume is reached.
- Similarly, if the volume is low, pressure is increased by 1-3 cm H_2O on each breath until the target is met or pressure limit is exceeded.

SPECIFIC FEATURES OF DELIVERED PRESSURE CONTROLLED BREATH

Decelerating Flow

Pressure controlled breaths are delivered with a decelerating flow. A servo mechanism regulates the inspiratory pressure on a breath to breath basis to achieve the target tidal volume. This mode is helpful to ventilate patients in whom the compliance and airway resistance are likely to change frequently.

Automatically Adjust the Inspiratory Pressure Control Level

In this control mode, the ventilator will automatically vary the inspiratory pressure control level, according to the mechanical properties of the lung/thorax, within a few breaths, to deliver preset target volumes. The ventilator will automatically, breath by breath, adapt the inspiratory pressure control level to changes in the mechanical properties of the lung/thorax to ensure that the lowest possible level is always used to deliver the preset tidal and minute volume.

Preset Tidal Volume and Frequency During the Preset Inspiratory Time

The breaths are delivered with preset tidal volume and frequency during the preset inspiratory time. The pressure is kept constant during the entire preset time. The inspiratory flow will be decelerating. The ventilator evaluates the exhaled volume on a breath by-breath basis and will reset the pressure support level as needed to guarantee the tidal volume.

FUNCTIONING OF PRVC (SUMMARY)

The flow pattern often decreases the peak inspiratory pressure (by approximately 5 to 7 cm H_2O) when compared with the same volume delivered in a volume control (constant flow) mode. Mean airway pressures may increase, averaging about 1 cm H_2O. Therefore, it overcomes the limitation of pressure support ventilation and mandatory minute ventilation by assuring a constant tidal volume, even with a lung with changing pulmonary mechanics.

When ventilators starts, the first breath is a test breath with an inspiratory pressure level of 10 cm H_2O (5 cm H_2O is set PEEP) above PEEP. After a few breaths the target volume will be achieved. Maximum available pressure level is 5 cm H_2O below preset level upper pressure limit (Fig. 35.3).

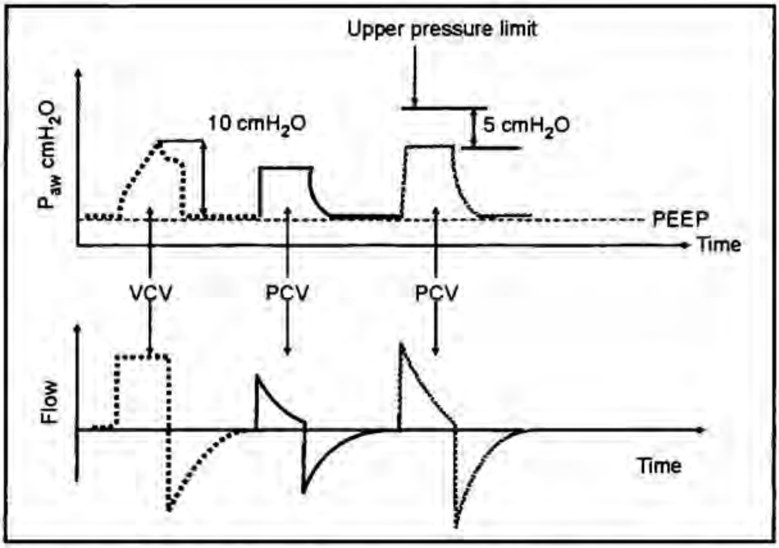


Figure 35.3: Test breath (volume controlled) followed by pressure controlled breath with decelerating flow

The inspiratory pressure support level changes breath by breath until preset tidal volume is obtained. When measured and preset tidal volumes correspond, the inspiratory pressure level remains constant (Fig. 35.4).

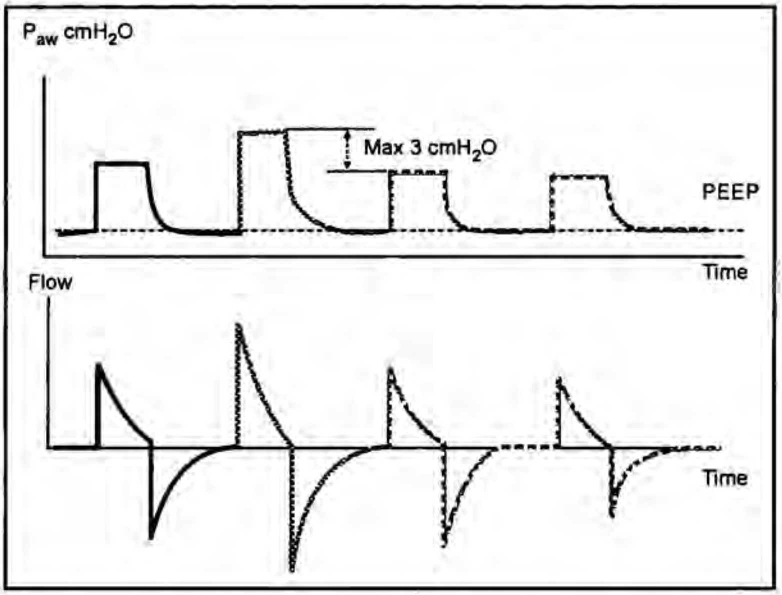


Figure 35.4: Adjusts the flow rate for the set tidal volume at or below the set maximum pressure

If the measured tidal volume increases above the preset, the pressure level decreases insteps of max 3 cmH_2O between consecutive breaths until the preset tidal volume is delivered. If the measured tidal volume decreases below the preset, the pressure level increases in steps of max 3 cmH_2O between consecutive breaths until the preset tidal volume is delivered. When measured tidal volume corresponds to preset value, the pressure level remains constant (Fig. 35.5).

When the ventilator regulates the inspiratory pressure the displayed set and measured tidal volumes can differ.

AIM OF MODE

- Deliver a preset tidal/minute volume with a preset frequency and with a constant pressure during the entire inspiration
- Deliver a decelerating inspiratory flow
- Delivered a controlled respiratory rate and I:E ratio
- Deliver a set tidal volume at the minimum pressure level necessary.

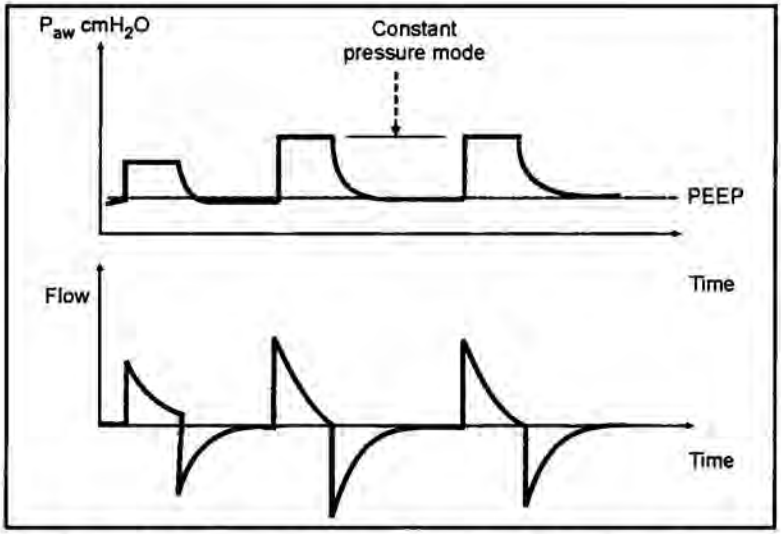


Figure 35.5: Adjusts the flow rate for the set tidal volume at or below the set maximum pressure

STRENGTH OF PRVC

PRVC gives you the clinical benefits of both volume controlled and pressure controlled ventilation, i.e. volume guarantee and ventilation with low inspiratory pressure but at the same time takes away the major disadvantage. PRVC diminishes the need for sedation and its effects on the circulation.

- Volume Controlled Ventilation
  During volume control the preset volumes will be delivered despite changes in lung/thorax mechanical properties. However, peak airway pressure can rise to high levels and this can interfere with the circulation, can increase the risk of barotrauma, and furthermore, can worsen lung parenchymal injury.
- Pressure Controlled Ventilation
  During pressure control the tidal volume is delivered with a decelerating flow, keeping inspiratory pressure constant during the entire inspiratory time. This improves gas distribution within the lungs, lower set peak inspiratory pressure giving rise to less circulatory interference and fewer adverse effects on lung parenchyma. The disadvantage is that there is no ventilation guarantee despite changes in mechanical properties.

WEAKNESS OF PRVC

- Minor for controlled mode
- No automatic shift to VS if patient triggers.

BENEFITS

- Inspiratory pressure always adapts to changes in lung/thorax mechanical properties
- It gives you a guaranteed tidal volume but minimize barotrauma. Some authors feel this is a friendlier mode for awake patients
- Lower peak inspiratory pressure and lower pressure swings
- Reduced cardiovascular interference
- Improved gas distribution with the decelerating flow
- Less need for sedation
- Shorter time spent in ICU
- Can be used on all patient's populations.

USES

- Patient with no breathing capacity or in whom you want to control the ventilation completely
- In whom you want a ventilation guarantee and ventilation with low inspiratory pressures and minimal pressure swings.
- In whom you want to have the same ventilation despite dynamic changes in mechanical properties of the lung/thorax
- In whom you want to use a decelerating flow.
- In patients with high airway pressure but can be used in any patient.

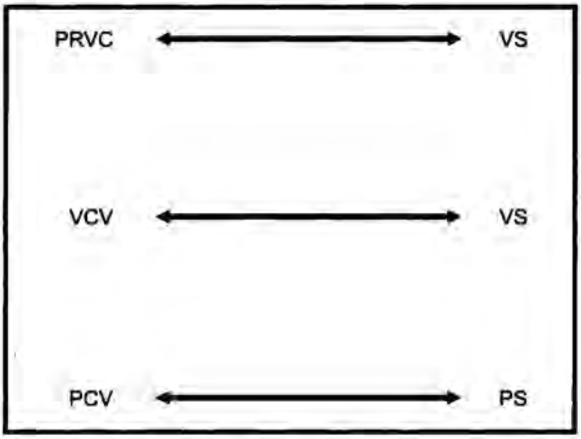
This approach is also most useful for patient with an inappropriate decrease in delivered airway pressure when the patient's inspiratory demand is increased as a result of increased stress (e.g. hypoxaemia, increased temperature, pain, anxiety).

AUTOMODE

Automode is present in Drager Evita & Siemens 300A. The ventilator continuously adapts to the patient's breathing capability and allows the patient, to interact with the ventilator in a better way. The ventilator automatically shifts between controlled ventilation, supported ventilation and spontaneous ventilation. Each controlled ventilation mode has a corresponding support mode.

Automode is a functionality where the ventilator adapts to the patient's varying breathing capacity and automatically shifts between a control mode and support mode using a fixed combination of variation modes. Automode is not possible in non-invasive ventilation (NIV). There are three different combinations depending on the mode installed. This is similar to PRVC but additionally allows the patient to breathe spontaneously at the upper and lower pressure levels in a manner similar to BIPAP.

The automode is a combination of a machine triggered with a patient triggered (spontaneous) mode, e.g. PRVC combined with VS, PCV with PS, VCV with VS. As the patient takes two triggered breaths, the mode automatically cycles into the spontaneous mode with the same settings.



The ventilator starts in control mode and operates according to the volume control, PRVC or pressure control mode. If the patient triggers breath, the ventilator will turn to support mode, to encourage the patient's respiratory drive.

If the patient is breathing adequately:

- *Volume control ↔ Volume support*

  The ventilator uses the plateau pressure in the volume controlled breath as a reference pressure for the first volume supported breath. In volume support the ventilator adjusts the inspiratory pressure level breath by breath to assure the preset target volume.

- *PRVC ↔ Volume support*

  The first supported breath delivered to the patient has the same pressure level as the preceding PRVC breath.

- *Pressure control ↔ pressure support*

  In this combination of automode, pressure control level (PC above PEEP) and pressure support level (PS above PEEP) can be adjusted to regulate the pressure level. In pressure support the ventilator assures that the preset inspiratory pressure level is maintained constantly during the entire inspiration.

If the patient is not breathing adequately and exceeding manually set trigger time out limit without sufficient patient effort will cause:

- In volume support; a PRVC or volume controlled breath will be delivered according to the selected Automode functionality.
- In pressure support; a pressure controlled breath will be delivered. Adaptation of trigger time out limit:

The ventilator initially adapts with dynamic trigger time out limit. This means that for the spontaneously breathing patient, the trigger time out limit increases successively until the set trigger time out limit is reached.

CHAPTER

36

Intrinsic Positive End-expiratory Pressure

INTRODUCTION

Auto-PEEP or intrinsic PEEP is defined as an unintentional PEEP that occurs when a new inspiratory breath is delivered before expiration has ended, in a patient on ventilatory support. Due to the dynamic effects of lung mechanics (resistance, compliance and closing volume) and the adjustment of parameters of ventilation, the intrinsic PEEP differs from the PEEP in the airways, i.e. extrinsic PEEP.

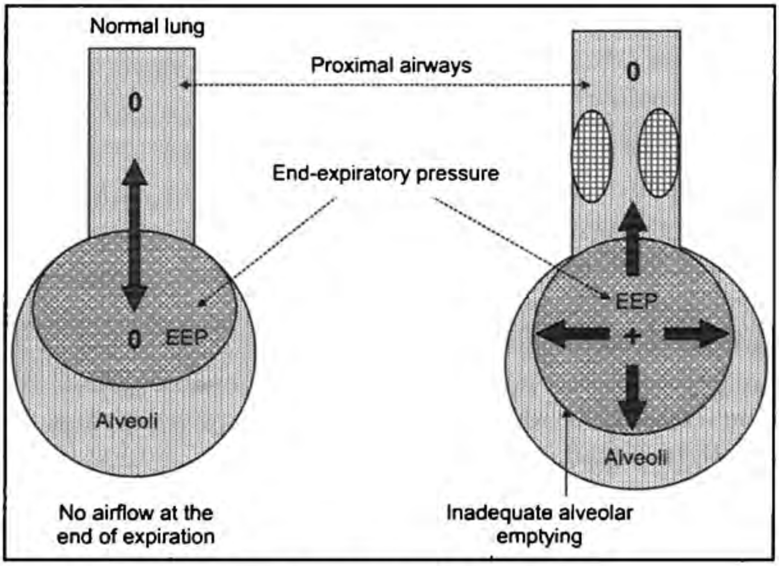


Figure 36.1: Pathogenesis of intrinsic PEEP

Pathogenesis of Intrinsic PEEP (Fig. 36.1)

Under normal circumstances, there is no airflow at the end of expiration and thus end-expiratory pressure is the same in the alveoli and proximal airways. The features of intrinsic PEEP are produced by inadequate alveolar emptying.

The presence of airflow at end-expiration creates a pressure drop from the alveolus (P_{alv}) to the proximal airways (P_{prox}).

Features of Intrinsic PEEP Produced by Inadequate Alveolar Emptying (Fig. 36.2)

The proximal airway pressure returns to zero at end-expiration. Due to inadequate emptying, the positive pressure present in the alveoli is not evident on the proximal airway pressure tracing (which shows zero), though alveolar pressure remains positive at end of expiration: Hence the term 'occult-PEEP.'

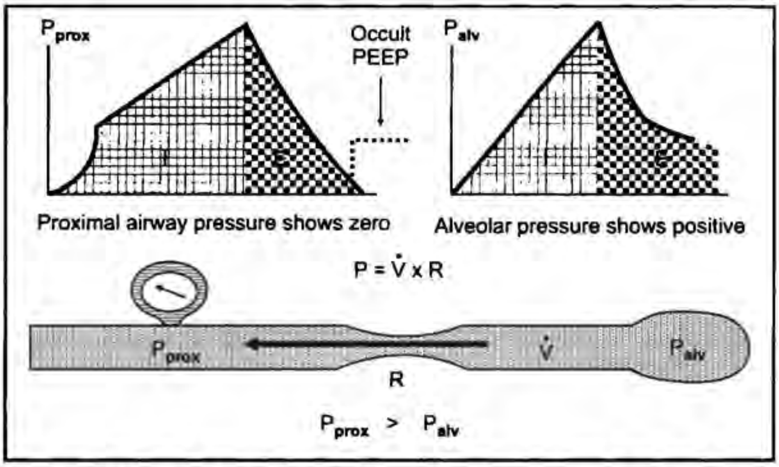


Figure 36.2: Features of intrinsic PEEP produced by inadequate alveolar emptying

Intrinsic PEEP or auto-PEEP is the actual end-expiratory pressure inside the lungs after end expiration when patient is on ventilator in with IPPV/CPPV mode.

MONITORING OCCULT PEEP

If complete expiration of a lung area is not possible within the time available, a residual volume remains in this area (trapped volume), giving rise to intrinsic PEEP, which is "residual pressure" during incomplete expiration. Auto-PEEP cannot be detected by observing the end-expiratory pressure either on the pressure time waveform or on the ventilator pressure monitor. The following methods have been used to monitor occult PEEP.

1. **Clinical Examination**

   Auto-PEEP may be assumed to be present when during auscultation of the lungs; inspiration commences before exhalation is complete.

2. **Monitor Flow Waveform (Fig. 36.3)**

   When flow waveforms are being monitored, the expiratory flow waveform does not return to baseline and the inspiratory flow starts from below the baseline.

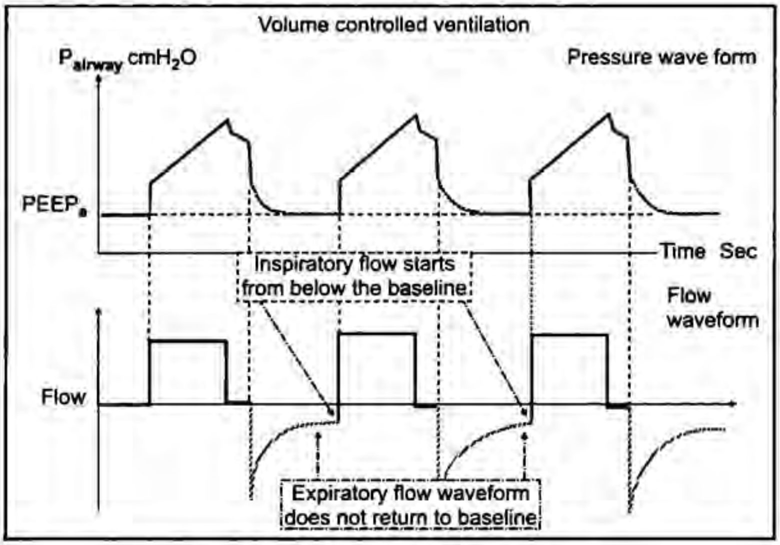


Figure 36.3: To monitor flow waveform

3. **Expiratory Hold Manoeuvre**

   If an expiratory hold manoeuver is performed, the end-expiratory pressure can be seen to rise to the total PEEP which is present in the system. The difference between the set–PEEP and the total PEEP is the auto-PEEP.

   Auto-PEEP = Total PEEP – Set PEEP

Normal Exhalation with no Air Trapping (Fig. 36.4)

Ventilator system during normal exhalation with no air trapping, no auto-PEEP and with the manometer reading zero.

Air Trapped in the Lung but not Recorded (Fig. 36.5)

The manometer still reads zero (ambient) because the exhalation valve is open to room air but there is 15 cm H_2O of air trapped in the lung.

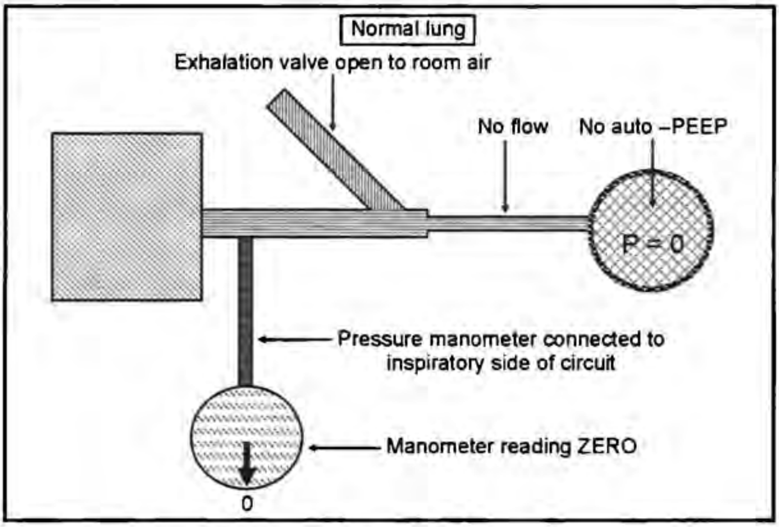

Normal lung
Exhalation valve open to room air
No flow
No auto –PEEP
P = 0
Pressure manometer connected to inspiratory side of circuit
Manometer reading ZERO
0

Figure 36.4: Normal exhalation with no air trapping

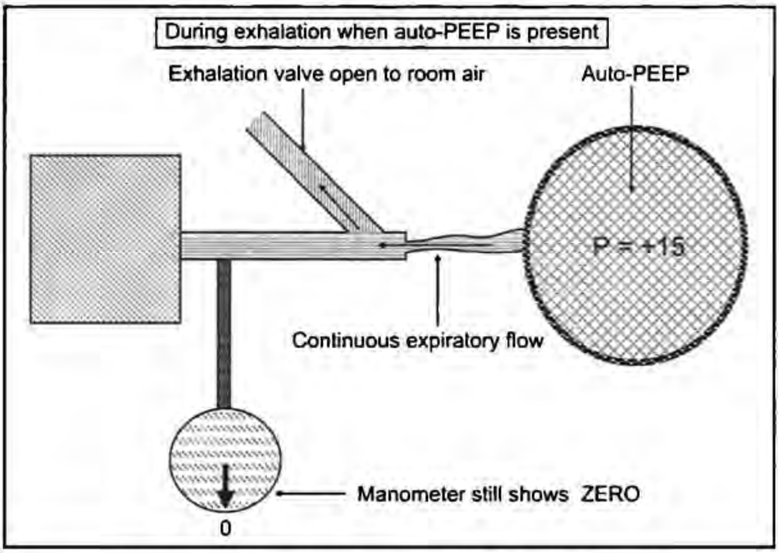

During exhalation when auto-PEEP is present
Exhalation valve open to room air
Auto-PEEP
P = +15
Continuous expiratory flow
Manometer still shows ZERO
0

Figure 36.5: Air trapped in the lung but not recorded

End-expiratory Occlusion Test

During controlled ventilation (i.e. when the patient is completely passive), occult-PEEP can be detected by occluding the expiratory tubing at the end of expiration. Measurement can only be carried out in the IPPV or IPPV/Assist ventilation modes.

Pressure equalisation (Fig. 36.6): When the exhalation valve is closed and inspiratory flow is stopped at end-exhalation and prior to the next breath, so that it is impossible for gas either to flow into the breathing system from inspiration or escape from it.

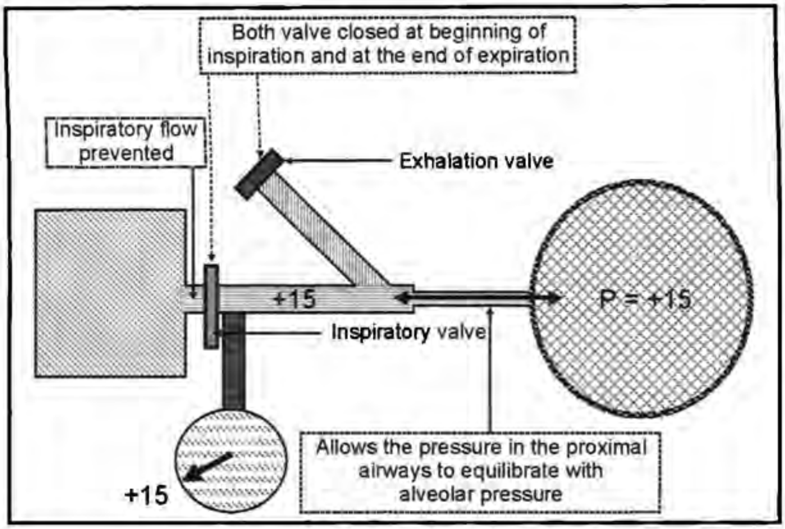

Both valve closed at beginning of inspiration and at the end of expiration
Inspiratory flow prevented
Exhalation valve
+15
P = +15
Inspiratory valve
+15
Allows the pressure in the proximal airways to equilibrate with alveolar pressure

Figure 36.6: Pressure equalisation

This manoeuvre blocks airflow and allows the pressure in the proximal airways to equilibrate with alveolar pressure. During this closed phase, pressure equalisation takes place between the lung and the breathing system.

- *De-pressurisation (Fig. 36.7):*
  When ventilator opens the expiratory valve and measures expiratory flow which corresponds to the volume trapped (V_{trap}) in the lung.

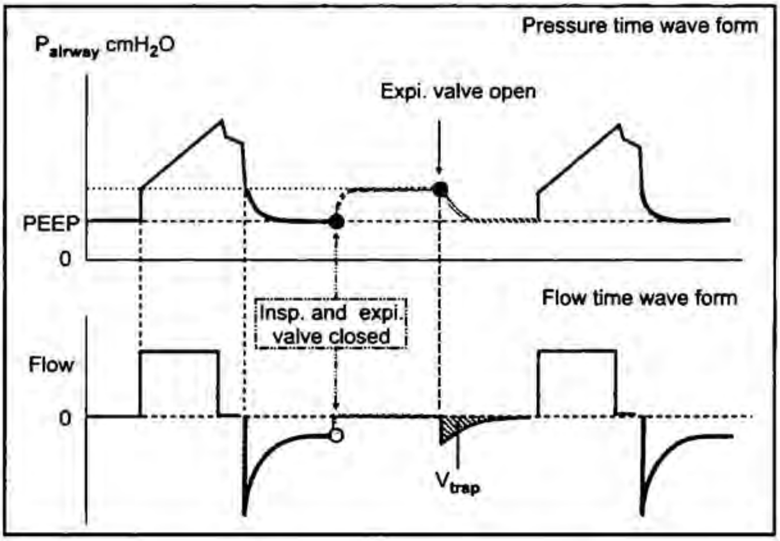


Figure 36.7: De-pressurisation after opening expiratory valve

A manometer will be able to read the approximate auto-PEEP level in the lungs and circuit. Then the lung is de-pressurised to PEEP. Thus, a sudden rise in proximal airway pressure with end-expiratory occlusion is evidence of occult-PEEP.

PRECAUTIONS

To measure the level of occult PEEP, the occlusion must be timed to the very end of expiration. Expiratory flow can persist until the end of expiration, so occlusion performed before end-expiration will overestimate the prevalence and magnitude of occult-PEEP.

CONSEQUENCES OF OCCULT PEEP

1. *Underestimation of thoracic compliance:*
   Occult-PEEP increases both the peak and plateau pressures, i.e. end-inspiratory pressure in the proximal airways. The increase in plateau pressure is misinterpreted as a decrease in the compliance of the lungs and chest wall when occult-PEEP goes undetected. This failure to detect occult-PEEP results in an underestimation of thoracic compliance.
2. *Adjust plateau pressure for compliance measurement:*
   When respiratory compliance is being monitored the level of occult PEEP should be subtracted from the measured plateau pressure. This adjusted plateau pressure should be used for the compliance calculation.
3. *Increased pressure measurement in CVP:*
   It can increase pressures measured by central venous or pulmonary artery catheters. It is necessary to exclude occult-PEEP if there is increase in pressures.
4. *Increased work of breathing:*
   If auto-PEEP is present, WOB is increased because the level of auto-PEEP in the lung at end-expiratory phase must be first overcome before inspiratory negative pressure can be used to reach the sensitivity setting.

SENSITIVITY SETTING TO TRIGGER BREATHE IN PRESENCE OF AUTO-PEEP

Under normal conditions, a mechanical breath is initiated when the inspiratory pressure reaches the sensitivity setting of the ventilator.

1. *Without auto-PEEP (Fig. 36.8):*
   When the normal end-expiratory pressure is 0 cm H_2O the pressure gradient to trigger a mechanical breath is 2 cm H_2O (from 0 cmH_2O to 2 cmH_2O).

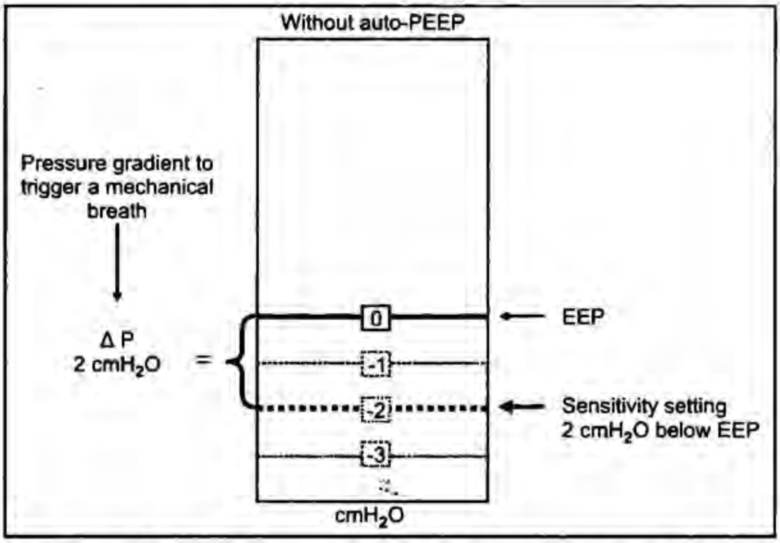


Figure 36.8: When the normal end-expiratory pressure is 0 cmH_2O

2. *With auto-PEEP of 6 cmH_2O (Fig. 36.9):*
   The pressure gradient from auto-PEEP to sensitivity setting is 8 cmH_2O; it increases work of breathing for the patient to trigger the ventilator for mechanical breath.

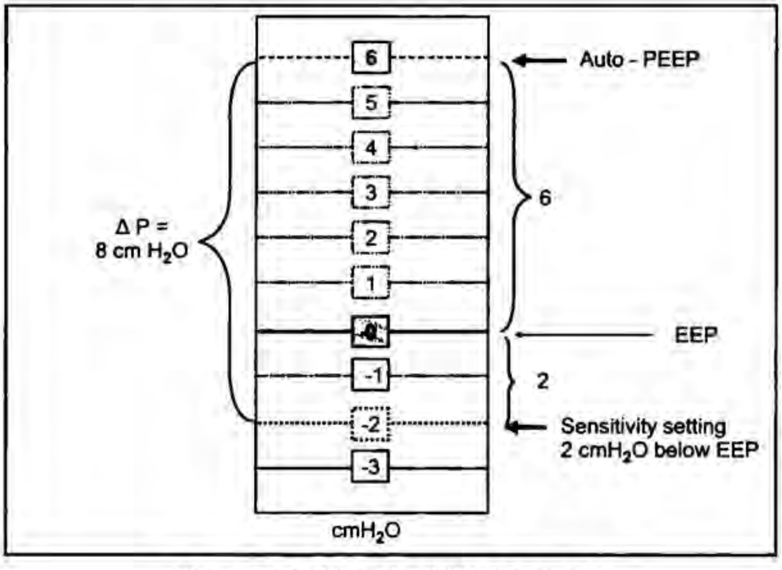


Figure 36.9: With auto-PEEP of 6 cmH_2O

3. *PEEP compensation in presence of auto-PEEP (Fig. 36.10):*
   In these patients extrinsic PEEP is set at 5 cmH_2O and trigger sensitivity is 2 cmH_2O. Pressure gradient to trigger a mechanical breath

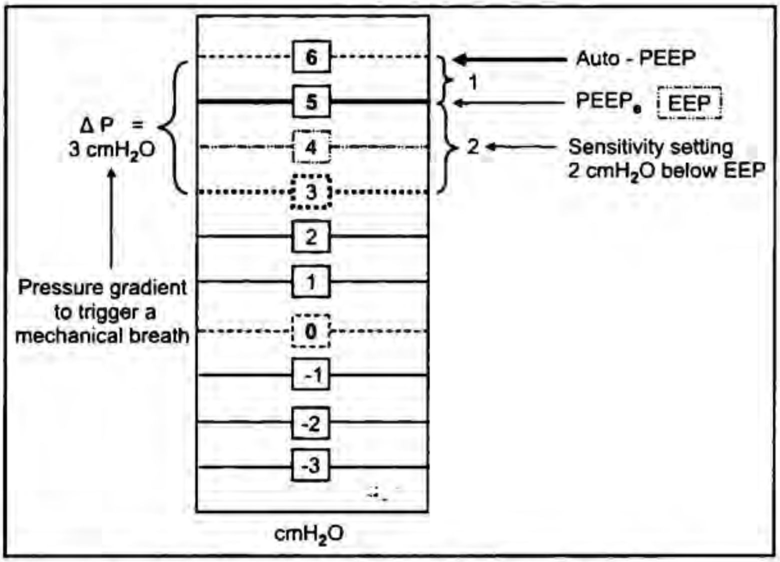

ΔP = 3 cmH₂O
Pressure gradient to trigger a mechanical breath
6
5
4
3
2
1
0
-1
-2
-3
cmH₂O
Auto - PEEP
1
PEEPₑ
EEP
2
Sensitivity setting 2 cmH₂O below EEP

Figure 36.10: PEEP compensation in presence of auto-PEEP

drops to 3 cmH_2O instead of 8 cmH_2O. 1 cmH_2O of pressure drop to bring auto-PEEP from 6 cmH_2O to 5 cmH_2O plus 2 cmH_2O to reach the preset sensitivity level. Trigger sensitivity is below end expiratory pressure. In the case of application of extrinsic PEEP, it is end expiratory pressure. That is why pressure drop is only 3 cmH_2O.

SETTING OF TRIGGER

1. Once the patient is allowed to trigger the ventilator, the cautious addition of PEEP (generally <10 cmH_2O) may narrow the difference between alveolar and auto-PEEP making the ventilator easier to trigger.
2. Extrinsic PEEP can be used to reduce the effects of auto-PEEP. The level of PEEP used to counter the effects of auto-PEEP should be kept below 85% of the measured auto-PEEP level.

PREDISPOSING FACTOR FOR OCCULT-PEEP

- Disease associate factor that promote hyperinflation.
- Airways obstruction, e.g. Asthma, COPD. The dynamic hyperinflation is other term usually reserved for PEEP produced by obstructive airway disease.
- ARDS
- Patient > 60 years of age
- Use of a small-sized endotracheal tube.

VENTILATOR FACTORS THAT PROMOTE HYPERINFLATION LEADING TO AUTO-PEEP

Trying to Maintain Normocarbia in Patients with Severe Bronchospasm

If ventilation is used with high respiratory rates and large tidal volumes or to decrease peak airway pressure, low peak inspiratory flow rates were used can result in development of high auto-PEEP levels.

Increased Expiratory Flow Resistance

There is increased expiratory flow resistance in a patient with asthma, or exacerbation of COPD. If an adequate expiratory time has not been given, inspiration commences before expiration is complete, leading to dynamic hyperinflation and auto-PEEP. Airway resistance can also increase during fibreoptic bronchoscopy through the endotracheal or tracheostomy tube in a patient receiving controlled ventilation. The fibrescope occupies the lumen of the tracheal tube, compromising its lumen and thus increasing airway resistance.

Increased inspiratory Time

Oxygenation is improved during inverse ratio ventilation and it is due to an increase in mean airway pressure and the inspiratory time. Inspiratory time is deliberately set greater than the expiratory time, (e.g. 1:E ratios 2:1, 3:1) leading to auto-PEEP.

Low Peak Inspiratory Flow Rates

Low peak inspiratory flow rates can prolong the inspiratory time. In patients with bronchospastic conditions, during volume-controlled ventilation high peak airway pressures are often observed. Attempt to reduce peak airway pressure by reducing the peak inspiratory flow rate can lead to increase in auto-PEEP.

VENTILATORY STRATEGIES IS TO LIMIT END-EXPIRATORY ALVEOLAR VOLUME AND PRESSURE

The best method to prevent or reduce hyperinflation and occult-PEEP is to promote alveolar emptying during expiration.

Improve Ventilation (Fig. 36.11)

Ventilation can be improved by using bronchodilators which reduce air trapping.

Small Tidal Volume

Tidal volume is decreased to 8-10 ml/kg, to decrease the volume of gas that must be exhaled on each breath.

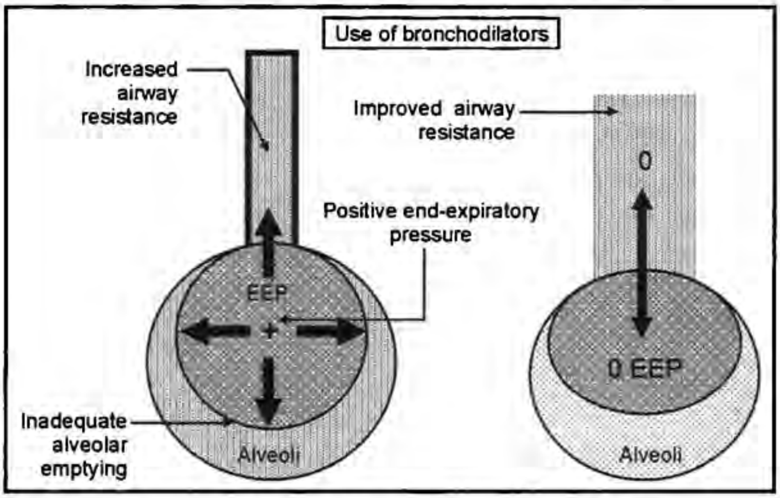

Use of bronchodilators
Increased airway resistance
Improved airway resistance
0
Positive end-expiratory pressure
EEP
+
0 EEP
Inadequate alveolar emptying
Alveoli
Alveoli

Figure 36.11: Bronchodilators improves ventilation

Reduce Respiratory Rate (Fig. 36.12)

The respiratory rate is decreased to 8-10/minute to reduce the minute ventilation and to increase expiratory tidal volume.

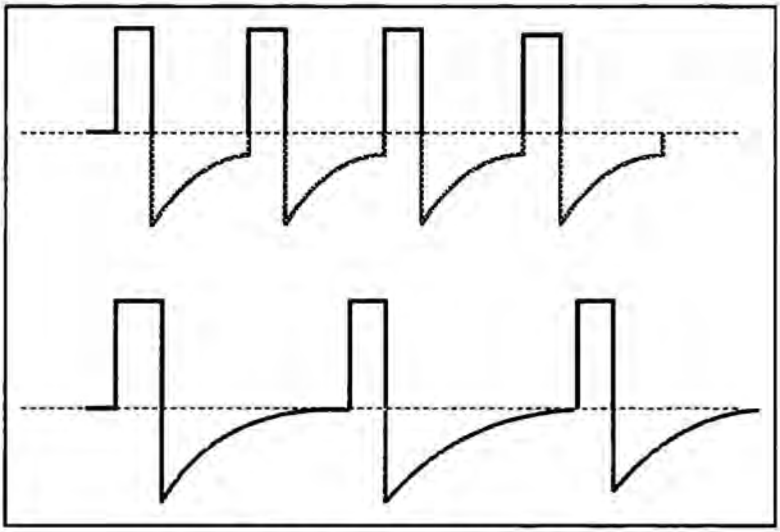


Figure 36.12: Reduction of respiratory rate

Prolonged Expiratory Time (Fig. 36.13)

Increasing the flow rate or reducing the tidal volume and/or respiratory rate, expiratory time can be prolonged.

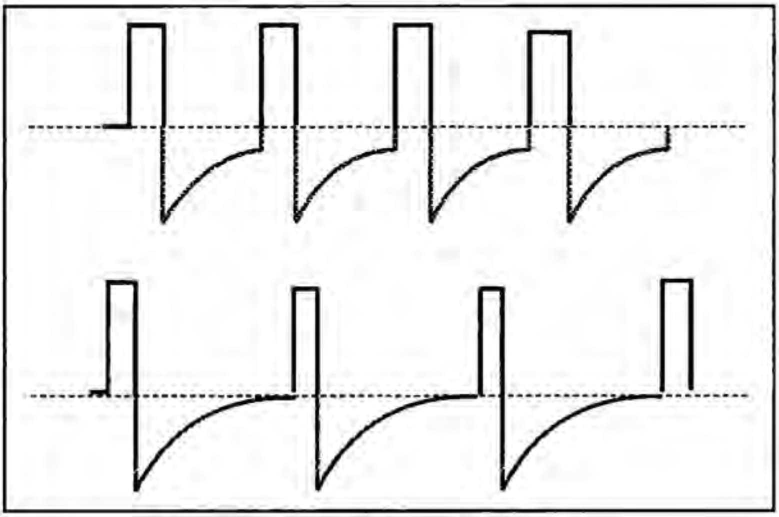


Figure 36.13: Prolongation of expiratory time

Higher Inspiratory Flow Rate (Fig. 36.14)

A higher inspiratory flow rate is beneficial since it will decrease inspiratory time and increase expiratory time. A decelerating waveform of inspiratory flow may help evenly distribute the tidal volume.

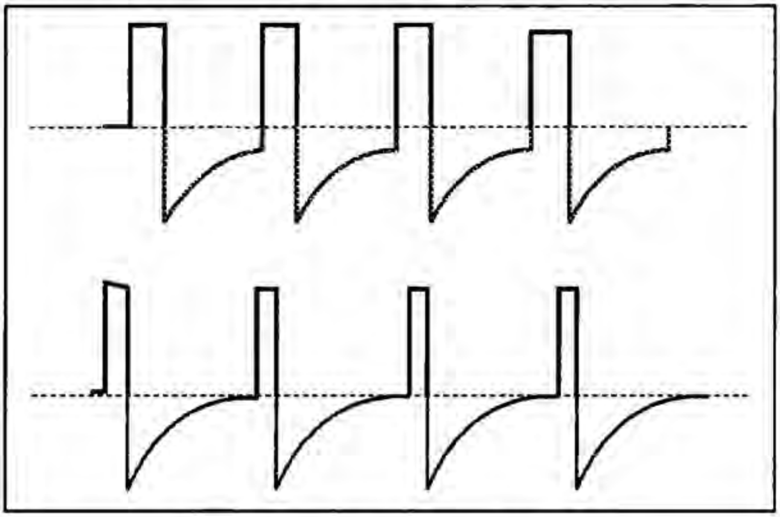


Figure 36.14: Higher inspiratory flow rate

ADVERSE EFFECTS OF AUTO-PEEP

Alveolar Rupture

Occult-PEEP is manifestation of hyperinflation; Hyperinflation-induced alveolar rupture (volutrauma) and pneumothorax are particularly concerns in ventilator-dependent patients.

Haemodynamic Compromise

Auto-PEEP and externally applied PEEP acts in the same way as to increase intrathoracic pressure and interfere with venous return to the heart. The increase in total PEEP (total PEEP = set PEEP + auto-PEEP increases the mean intrathoracic and mean airway pressures, leading to decreased cardiac output and hypotension.

Precautions

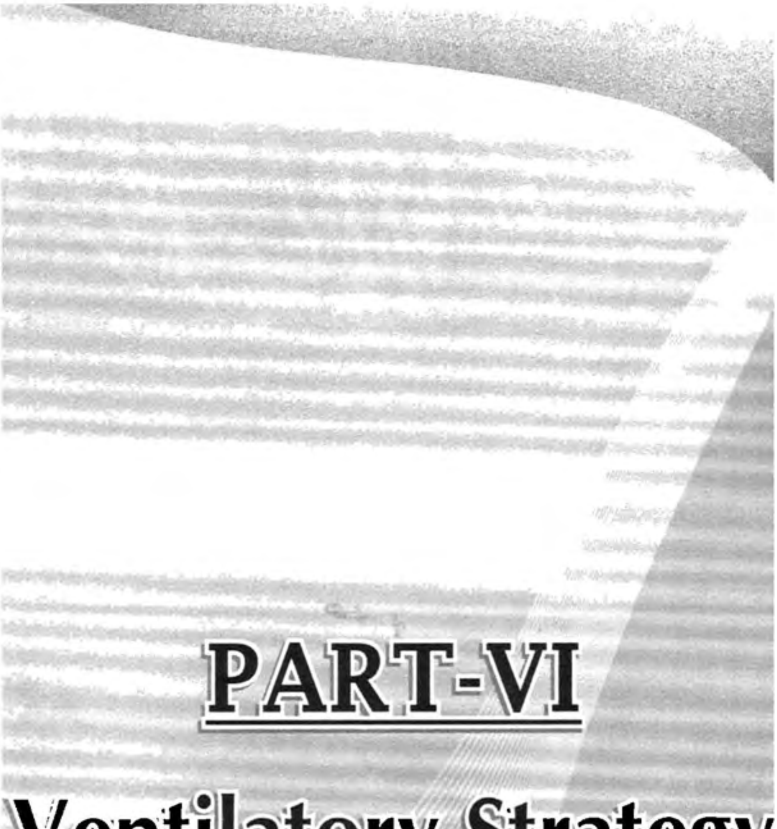
Hypotension due to auto-PEEP may develop within 1-2 min, due to a rapid breathing pattern and may be reversed as quickly by stopping or slowing the ventilator. Development of auto-PEEP must be considered in the differential diagnosis of haemodynamic compromise in a mechanically ventilated patient especially in the presence of obstructive airway diseases.

Increased Work of Breathing

In a spontaneously breathing patient requiring assisted ventilation, the patient has to generate a negative intrathoracic pressure to initiate inspiratory flow from the ventilator. Patients who have auto-PEEP must first generate a negative intrathoracic pressure that equals auto-PEEP value, before they can generate additional negative airway pressure needed to trigger the ventilator. This requires not only greater effort but it increases the energy the patient must generate to trigger the ventilator.

Auto-PEEP markedly increases the work of breathing by two mechanisms:

- The hyperinflation places the lungs on a flatter portion of their pressure-volume curve, in a region of lower compliance, so higher pressures are needed to inhale the tidal volume. (To appreciate this effect, take a deep breath, and then try to breathe in further). This higher pressure posing as an inspiratory threshold load on the respiratory muscle, difficult for the patient to trigger the ventilator during any assisted mode of ventilation, leading to patient-ventilator dysynchrony, which increases work of breathing giving rise to respiratory muscle fatigue.
- The hyperinflation also flattens the diaphragm and when this occurs, the diaphragm muscle must generate a greater tension to achieve a given change in thoracic pressure. The increased WOB with occult PEEP can impede efforts to wean from mechanical ventilation. Patient uses considerable muscular effort but without triggering the ventilator and may prolong ventilator dependence.



PART-VI

Ventilatory Strategy

CHAPTER

37

Applications of Basic Principles in Respiratory Failure

Respiratory failure is the inability to maintain the normal homeostasis of arterial blood gases, leading to oxygen tension in arterial blood (PaO_2) of 60 mmHg or less and the carbon dioxide tension in arterial blood ($PaCO_2$) is 50 mmHg or more.

TYPES OF RESPIRATORY FAILURE

Acute Ventilatory Failure

Acute ventilatory failure occurs when alveolar ventilation cannot adequately remove carbon dioxide produced by cell metabolism, via the lungs. Ventilator failure always results in a rise in $PaCO_2$ and a fall in PaO_2. Hypercapnia is the feature of ventilatory failure and often termed as hypercapnic respiratory failure. When carbon dioxide production exceeds its removal, respiratory acidosis results. Hypoxaemia can be the secondary complication of ventilatory failure but this type of hypoxaemia usually responds well to oxygen therapy. The degree of hypoxaemia corresponds to the severity of ventilatory failure without supplemental oxygen.

Physiopathology of Ventilatory Failure

The carbon dioxide produced by tissue metabolism (VCO_2) is removed by the lungs. Normally the alveolar carbon dioxide tension $PACO_2$ and the arterial carbon dioxide $PaCO_2$ are maintained around 40 mmHg by adjusting alveolar ventilation. Increase in tissue metabolism is met by proportional increase in alveolar ventilation.

Alveolar ventilation (V_A) or effective ventilation is defined as that proportion of tidal or minute volume which takes part in gas exchange. The other part, in which no or imperfect gas exchange takes place is defined as dead space (V_D). A normal value of alveolar ventilation is about 3.5 to 4.5 lit/min in adults.

$$V_A = f\ (V_T - V_D)$$

Its importance lies in the fact that it is the pulmonary factor controlling the excretion of carbon dioxide by the lungs and it is directly related to the tidal volume, physiological dead space and respiratory rate.

$$PACO_2 = PaCO_2 = \frac{\dot{V}CO_2 \text{ (Carbon dioxide production in ml/min)}}{V_A}$$

$$PACO_2 = PaCO_2 = \frac{\dot{V}CO_2}{\dot{V}_E \text{ (expired minute ventilation)} - \dot{V}_D}$$

A rise in $PaCO_2$ due to ventilatory or hypercapnic respiratory failure can occur under following condition:

- *A fall in alveolar ventilation, this could be due to:*
  - A fall in minute ventilation
  - A rise in physiological dead space, without concomitant increase in minute ventilation
- *A rise in carbon dioxide production* without a proportionate increase in alveolar ventilation.

Important Causes of Hypercapnic Ventilatory Failure (Hypoventilation)

1. *Decrease in minute ventilation in normal lung*

   Hypoventilation in normal lungs is mainly seen in patients with a depressed ventilatory drive or in neuromuscular disease.
   - A depressed ventilatory drive
     - Poisoning—narcotics, antidepressants, sedatives
     - Neurologic dysfunction—coma from head injuries stroke, increase intracranial tension, encephalitis
   - Neuromuscular disease—tetanus, myasthenia gravis, snake bite poisoning, G.B. syndrome, acute poliomyelitis.
   - Large airway obstruction—tracheal/Subglottic stenosis.

   In the above conditions the lungs are normal to start with however if conditions are not managed properly, secretion accumulates in the lung producing areas of atelectasis. As a result of these changes within the lungs, gas exchange is further impaired due to ventilation perfusion imbalance.

2. *Decrease in minute ventilation and/or increase dead space ventilation, i.e. abnormal lung*
    - Severe airway obstruction – acute severe bronchial asthma, exacerbation in COPD
    - Thromboembolism
    - Terminal stages of restrictive lung disease
3. *Increased production of carbon dioxide*
    - High fever, uncontrolled seizers, tetanus
    - Respiratory muscle fatigue.

Hypoxaemic Respiratory Failure

Acute hypoxaemic respiratory failure results from poor gas exchange of oxygen within the lungs leading to a low PaO_2 (partial pressure of oxygen in arterial blood). The PaO_2 may be normal or even less than normal. Reduced/inadequate oxygenation (PaO_2) will increase alveolar arterial oxygen difference ($A\text{-}aDO_2$). It is due to all disorder of alveolar-capillary membrane. Oxygenation failure is defined as severe hypoxaemia ($PaO_2 < 40$ mm Hg) that does not respond to moderate to high levels (50-100%) of supplemental O_2.

Physiopathology of Hypoxaemic Failure

The normal alveolar ventilation (V_A) is about 4-5 lit/min and the normal perfusion (Q) around 5 lit/min. In the normal lung, there are regional differences in ventilation perfusion ratios, giving rise to V_A/Q ratio approximately is 0.8-1.

Acute hypoxaemic respiratory failure occurs when:

- Ventilation perfusion mismatch, characterised predominantly by low ventilation perfusion ratio in the lung.
- Significant increase in the right to left shunt due to perfusion of atelectatic alveoli.
- Combination of ventilation perfusion mismatch and increase in shunt.
- Marked decrease in diffusion of oxygen across to alveolar capillary membrane.

Important Causes of Hypoxaemic Respiratory Failure

- *Obstructive airway disease*
    - Exacerbation in COPD
    - Acute severe bronchial asthma
- *Restrictive lung disease*
    - Acute pulmonary oedema
    - Acute pulmonary infection

  - Pulmonary atelectasis
  - Acute interstitial disease
- *Acute thromboembolism*
- *Trauma to the chest* – flail chest.

Effects of Increased FiO_2 on

- *V/Q abnormalities*
  Increase in FiO_2 rapidly produces an increase in the PaO_2 and the oxygen content of blood. Thus, hypoxaemia of low V/Q is easily corrected.
- *Shunt*
  The treatment of hypoxaemia due to shunt is unpredictable. The greater the shunt, the poorer the response in PaO_2 to arise in FiO_2.

A COMBINATION OF ACUTE VENTILATORY AND HYPOXAEMIC FAILURE CAN GIVE RISE TO RESPIRATORY MUSCLE FATIGUE DUE THE SEVERE LUNG PATHOLOGY SUCH AS:

Failure of Oxygen Transport

A satisfactory gas exchange at the alveolar level must be accompanied by an adequate transport of oxygenated blood to the tissues. This requires an adequate cardiac output, a normal oxygen content of arterial blood and good tissue perfusion. A low output state from any cause can indirectly lower the PaO_2.

- It increases the physiological dead space
- It increases oxygen extraction from the blood by the tissues.
  Respiratory muscle fatigue—hypotension and shock lead to poor perfusion of respiratory muscles and quick fatiguability causing hypoventilation. This increases ventilation perfusion inequalities and can further reduce PaO_2. Excessive work performed by the muscle of respiration leads to fatigue. Increasing fatigue results in increasingly poor function and hypoventilation, and in the end stage the patient stops breathing, e.g. acute severe asthma, low pulmonary compliance. The more critically ill the patient, electrolyte disturbances, unstable circulation, poor perfusion leads to easier and quicker fatigue in respiratory muscles.

Features of Respiratory Muscle Fatigue

- Complaints of fatigue in relation to breathing—serious complain
- Respiratory rate > 35/min is a cardinal symptom of respiratory muscle fatigue.
- Poor chest excursions, irregular breathing, apnoeic spells.

- Respiratory alternans – intercostal and diaphragmatic contraction alternates.
- Use of accessory breathing muscle, paradoxical respiratory movement, inspiratory intercostals recession.
- Agitation, confusion, increased drowsiness.
- Tachycardia, hypertension.
- A minute ventilation >10-12 lit/min – difficult to sustain for long time in critically ill patient, VC< 3 times tidal volume.
- Blood gas analysis— Only blood gas analysis allows precise evaluation of the extent and type of respiratory failure. PaO_2 and $PaCO_2$ are essential parameters for initiation and administration of ventilatory support.

Treatment of Respiratory Muscle Fatigue:

Respiratory muscle fatigue should be managed by resting respiratory muscles by mechanical ventilation. During the period of rest, the acutely depleted glycogen stores of the respiratory muscles are replenished, and lactic acid and other metabolites associated with muscle fatigue are washed out.

Failure of Oxygen Uptake

In cyanide poisoning, there will be deficient oxygen uptake at the cellular level inspite of normal ventilation, normal exchange of blood gases at the alveolar level and good oxygen transport (normal cardiac output). Cyanide inactivates intracellular enzyme (cytochrome oxidase) hampering intracellular respiration, in spite of normal PaO_2, SaO_2, CaO_2, and oxygen transport. The patient will die of tissue hypoxia. Septic shock and acute lung injury are also associated with failure of oxygen uptake.

CLINICAL FEATURES OF ACUTE RESPIRATORY FAILURE

Acute respiratory failure is a failure to adequately oxygenate the tissues. The oxygen supply and oxygen uptake of especially vital organs are of crucial importance.

Hypoxia

Hypoxia is the basic underlying feature in every patient with acute respiratory failure. Hypoxia is reduced oxygen in the body organs and tissues. It is important to understand that hypoxia can occur with normal $PaCO_2$. Hypoxia depresses cell function, produces metabolic acidosis and if severe leads to death. The presence of hypoxia (decreased oxygen in organs and tissues) may not always be apparent. The only sure way to detect hypoxia is by measuring the arterial PaO_2.

Hypoxaemia is reduced oxygen in the blood. Hypoxaemia is present when the oxygen level (e.g. PaO_2, SaO_2) is decreased in arterial blood. The PaO_2 is most often used to evaluate a patient's oxygenation status. Since PaO_2 reflects only the oxygen that is dissolved in the plasma, it does not represent all oxygen carried by the (haemoglobin and plasma). For precise assessment oxygen content should be used as it includes the oxygen combined with haemoglobin as well as oxygen dissolved in the plasma.

Compensatory Mechanism by Hypoxia

Compensatory mechanisms of hypoxia are easily recognized and are therefore of diagnostic value.

- Sympathetic stimulation—tachycardia, hypertension. In the absence of sympathetic activity (old and feeble patient, drugs affecting autonomic system), sympathetic response may not be seen or feeble.
- The myocardium has no oxygen reserve; hypoxia depresses myocardium leading to bradycardia, arrhythmias, increasing hypotension and ultimate circulatory failure.
- Stimulation of chemoreceptors (carotid body and aorta)—increase in respiratory rate. The increase in respiratory rate will not be seen if the respiratory centre is depressed or respiratory muscles are weak or paralysed.

Hypercapnia

The hypercapnia is clinically difficult or impossible to detect, therefore it is necessary to measure $PaCO_2$.

- Depressant effects on the cardiovascular system—producing generalised vasodilation (except in the pulmonary circulation), manifesting as cutaneous flushing, warmth, sweating and a bounding pulse.
- Depressant effect on central nervous system—leads to drowsiness, confusion, disturbance in behaviour, flap tremor.
- Stimulation of sympathetic nervous system—producing tachycardia and hypertension. It also stimulates the respiratory centre the increasing rate and tidal volume (unless depressed centre or decreased sensitivity to centre).
- Carbon dioxide narcosis—can produce metabolic cause of coma. The coma of carbon dioxide narcosis is associated with loss of the deep reflexes and urinary incontinence.

The pattern of symptoms is balance between the depressant action on the cardiovascular system and the stimulant effects on the sympathetic nervous system.

INDICATIONS OF RESPIRATORY SUPPORT

The indications of respiratory support are therefore based on two physiopathology mechanisms:

1. *Type I or hypoxaemic respiratory failure, i.e. inadequate oxygenation:* Where the patient is unable to meet the oxygen requirements of the body or is able to do so only at a very high cost that result in a haemodynamic and metabolic compromise.
2. *Type II or hypercapnic respiratory failure reduced CO_2 elimination:* Where the ventilatory pump has failed due to central respiratory depression, neuromuscular weakness, severe bilateral chest wall and pulmonary abnormalities or severe airway obstruction.

The decision to intubate and initiate mechanical ventilation has always been more complicated than it should be. Instead of the usual list of clinical and physiological indications for mechanical ventilation, the following simple rule should simplify the dilemma.

1. *Thinking of it:*
   There is a tendency to delay intubation and mechanical ventilation as long as possible in the hopes that respiratory support may be unnecessary. However, elective intubation carries fewer dangers and complications than emergency intubation. Thus delays in intubation create unnecessary dangers for the patient. When you see a patient if you think, the patient's condition is serious enough for intubation and mechanical ventilation then proceed without delay.
2. *Intubation is not an act of weakness:*
   It is advisable to establish control of the airways. There is wrong a tendency to view intubation as an act of cowardice. It is safer to intubate "prematurely" than to perform a "post-mortem" intubation.
3. *Endotracheal tubes are not a disease and ventilators are not an addiction*:
   The assumption that "once on a ventilator, always on a ventilator" is a misleading notion and should never influence the decision to initiate mechanical ventilation. Endotracheal tubes and ventilators do not create the need for mechanical ventilation; however cardiopulmonary and neuromuscular diseases do. Whenever in doubt, get control of the airway.

It must be kept in mind that access to the airway for removal of secretions or protection of the airway in patients with impaired airway reflexes is an indication for endotracheal intubation or tracheostomy and not necessarily for mechanical ventilation. Failure to ventilate or oxygenate adequately may be caused by pulmonary or non-pulmonary conditions, often leading to a combination of dead pace ventilation, V/Q mismatch, shunt, diffusion defect.

But for the ease of the patient management, they are separated into three distinct groups

1. *Depressed respiratory drive:*
   Depressed or insufficient respiratory drive may lead to a decrease in tidal volume, respiratory rate or both. These patients may have normal pulmonary function but the respiratory muscles do not have adequate neuromuscular impulses to function properly. Mechanical ventilation is used to support these patients until the cause of insufficient respiratory drive has been reversed.
2. *Excessive ventilatory workload:*
   When excessive ventilatory workload exceeds the patient's ability to perform ventilatory work mechanical ventilation is necessary.
3. *Failure of ventilatory pump:*
   Failure of the ventilatory pump is the structural dysfunction of the respiratory system which includes the lung parenchyma and respiratory muscles. If uncorrected, this condition leads to increased work of breathing and eventual ventilatory and oxygenation failure.

Traditional Goals of Mechanical Ventilation

- *Correct hypoxaemia* – PaO_2 - 100 mmHg and O_2 Saturation 100%
- *Correct hypercapnia* - $PaCO_2$ - 40 mmHg
- *Apply PEEP* only if oxygenation can not be maintained
- *Reduce work of breathing* and hence cardiac workload by providing rest to respiratory muscles which also reduces oxygen cost of breathing by giving muscle relaxants and sedation.

Clinicians all over the world have realised the deleterious effects of traditional goals of mechanical ventilation. They have also realised deleterious effects of supine position and reduced functional residual volume during mechanical ventilation, coupled with prolonged sedation and paralysis which promote alveolar atelectasis. Therefore, the goals of mechanical ventilation have been modified to reduce the risk of ventilator induced lung injury.

MODIFIED GOALS OF MECHANICAL VENTILATION

1. *Correct hypoxaemia, i.e. oxygenation*
   - Saturation < 90% with minimum FiO_2 , PaO_2- 60 mmHg
   - Recruit alveoli and keep alveoli patent
   - Titrate PEEP to keep end expiratory volume above lower inflection point and achieve best lung compliance
   - Titrate PEEP and PIP to maximize O_2 delivery (as high pressures will decrease venous return)

2. *Allow permissive hypercapnia*
    - Accept $PaCO_2 > 50$ mm Hg, pH < 7.2
    - Pressure preset ventilation
    - Limit tidal volumes-6-8 ml/kg
    - Avoid overdistension-Limit PIP < 35 cm H_2O and $P_{plateau}$- < 30 cm H_20
3. *Implement spontaneous breathing early*
    - Use newer modes like PSV, proportional assist ventilation, volume support,
    - Prolonged sedation and paralysis may do more harm than good.

HOW TO START MECHANICAL VENTILATION

Remember positive pressure ventilation is not physiological and harmful to the patient if not used wisely. It is not curative but supportive to buy time.

Evaluate Need for Ventilatory Support

- *Is the respiration clinically adequate or are any signs of impending failure present?*

  How does the patient look ? –fatigued, tired or comfortable.

  Shallow respiration, discordinated movement of the chest and the abdomen or use of accessory muscles excessively, respiratory rate less than 6/min or greater than 30 are signs of impending respiratory failure.

- *Is the airway adequate and patent?*

  Assess gag reflex, swallowing ability, pooling of saliva, nasal regurgitation, expiratory grunting. Patients with worsening muscle weakness, a secure airway may help prevent aspiration especially during sleep.

- *Is there sufficient respiratory muscle strength?*

  V.C. < 30 ml/kg — Inability to cough well
  V.C. < 20 ml/kg — Inability to sigh, which prevents atelectasis
  V.C. < 10 ml/kg — Inability to ventilate adequately

- *Is there adequate gas exchange?*

  A PaO_2 less than 60 mm Hg and/or $PaCO_2$ greater than 50 mm Hg indicates impending respiratory failure. $PaCO_2$ rises about 4-5 mm Hg/min of apnoea.

  ABG may be normal or show hypoventilation and/or hypoxaemia. Is the trend worsening or improving?

- *Is the patient haemodynamically stable?*

  A blood pressure of more than 90 mm Hg off inotropic support is indicative of a good cardiac pump function.

Providing Respiratory Support

- *Will oxygen meet your requirements?*
  Oxygen will be adequate only if muscle strength/VC is adequate and only if upper airway is not compromised. Otherwise hypoventilation and dangerous respiratory acidosis will supervene. Monitoring oxygen saturation alone in this situation may be misleading, as hypoventilation will not be picked up.

- *When to intubate?*
  Mechanical ventilation becomes necessary when spontaneous ventilation is inadequate for efficient gas exchange and the patient needs intubation. A patient with compromised airway, in a patient with progressive weakness where sooner or later invasive ventilation will be required, noninvasive ventilation should only be used as a bridge.

- *Can noninvasive ventilation be tried?*
  Noninvasive ventilation using a comfortable reasonably tight nasal or full face mask can be used in certain selected groups of patients. which can reduce the need for invasive ventilation. Certain basic requirements must however be met: Patient should be conscious. cooperative with intact airway, preferably haemodynamic stabile. Acute rise in CO_2 means to consider invasive technique. Patients with $pH < 7.1$ and $PCO_2 > 70$ mm Hg should not be considered for a non invasive technique. The Non - invasive technique can be used in acute exacerbations of COPD, sleep apnoea, acute hypoxaemic respiratory failure in the early stages, e.g. pulmonary oedema, bronchial asthma, early ARDS.

Guidelines to Start the Patient on the Ventilator

- Set the parameter after understanding what type of respiratory failure you are dealing. Choose the right time to start the ventilator
- *If in doubt, intubate first, think later*
  Check BP every 15 minutes for first hour or till the patient stabilised since the BP can fall severely.
- The setting should be determined by the physiopathology of acute respiratory failure needing mechanical ventilation. Neither fixed setting nor mode is dominated.
- First blood gases should be done after 30 to 60 minutes of starting the ventilator and then readjustments. Watch other systems as well.

CHAPTER

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Ventilatory Management of Severe Asthma

Asthma is defined as chronic inflammatory disorder of the airways which is characterised by acute generalised airway obstruction which is reversible in nature. This is due to hyper reactivity airways caused as a result of release of inflammatory mediators (e.g. histamine).

Life-threatening asthma is associated with a markedly increased airway resistance, pulmonary hyperinflation and high physiological dead space, which together lead to hypercapnia and risk of respiratory arrest. In the asthmatic patient, sudden asphyxial attacks which are acute in onset often triggered by airway irritants associated with profound bronchospasm with minimal mucous plugging called *'sudden death syndrome'*.

PATHOPHYSIOLOGY AND CLINICAL PRESENTATION OF AN ACUTE ASTHMA ATTACK

Airway Obstruction

The airway obstruction in asthma is widespread and uneven in distribution and mainly of smaller airways. Airway obstruction is due to a variety of reasons such as smooth muscle spasm, bronchiolar mucosal inflammation and oedema, thick tenacious mucous plugging of smaller airways. There is greater tendency for closure of the airway at higher lung volumes than lower in case of acute severe asthma attack. The increase in lung volume increases static transpulmonary pressure. This results in an increased outward radial traction on the airway, trying to keep airway open. The airway obstruction is reflected in decrease in forced expiratory volume in one seconds (FEV_1) and peak expiratory flow rate (PEFR).

Hyperinflated Chest

Increased lung volume will be seen clinically and radiologically by hyperinflated chest.

Physiological Changes of Increased Lung Volume

The increase in lung volume markedly increases residual volumes (RV), total lung capacity (TLC) and RV/TLC. During recovery of hyperinflation TLC decreases before there is increase in FEV_1 and PEFR.

Ventilation and Perfusion Imbalance

Ventilation and perfusion imbalance gives rise to gas exchange abnormalities and hypoxaemia. Severity of airway obstruction and hypoxaemia have a linear relationship due to increase in number of low ventilation perfusion units. PaO_2 below 60 mm Hg was usually associated with FEV_1 of < 0.5 lit. If airway obstruction is complete for some lung units then there is increase of right to left shunt which is an additional factor for hypoxaemia.

Respiratory Muscle Fatigue

The asthmatic patient due to hyperventilation tends to wash out CO_2 from the blood during an asthma attack. Consequently low $PaCO_2$ is a rule than exception in acute asthma and normal $PaCO_2$ in these patients implies respiratory muscle fatigue. A normal $PaCO_2$ in asthmatic patients is an alarming sign and should be closely monitored. At a later stage rising $PaCO_2$ and falling pH denotes exhaustion of respiratory muscles.

CLINICAL PRESENTATION DUE TO AIRWAY NARROWING

Respiratory failure from airflow obstruction is a direct consequence of a critical increase in airway resistance due to airway narrowing leading to two important mechanical changes:

Ventilatory Pump Failure—During Spontaneous Ventilation

During the early stage of an asthmatic attack, there may be a brief period of hyperventilation which may give rise to increase in minute ventilation. But during a later stage, minute ventilation decreases because of respiratory muscle overloading mainly due to overinflation and airflow obstruction giving rise to increase in work of breathing.

The work of breathing during severe asthma attack is estimated at 5-25 times that done by normal individuals at rest. The increased pressure required for maintenance of expiratory airflow may overload respiratory muscles producing a "ventilatory pump failure" with spontaneous ventilation. The ventilation produced by fatigued respiratory muscles is inadequate for gas exchange. Since it is more of a failure of ventilation and CO_2 removal it is also termed as *"hypercapnic respiratory failure"*.

Pulmonary Hyperinflation—During Mechanical Ventilation

During mechanical ventilation dynamic hyperinflation is initiated when there is insufficient time (when ventilated at high rates) for complete exhalation of delivered tidal volume. The next mechanical breath is delivered before expiration is completed and insufficient time is available for the lungs to empty to their resting volume due to narrowed airways.

- This results in an increase in end-expiratory lung volume and positive end-expiratory alveolar pressure (auto-PEEP).
- With subsequent breaths, there is a progressive increase in lung volume. This leads to an improvement in expiratory gas flow because of higher elastic recoil pressure (manifested as auto-PEEP or called as air trapping) and increase in airways diameter, allowing the entire tidal volume to be exhaled.
- 100% of patients ventilated for acute severe asthma or exacerbation of chronic obstructive lung disease have measurable levels of auto-PEEP that may be as high as 15-20 cm H_2O.

Therefore, dynamic hyperinflation is an adaptive process which enhances expiratory flow in the case of airway obstruction but also increases risk of complications related to alveolar overdistension. The increased volumes of trapped gas give rise to a vicious cycle of further hyperinflation, barotrauma and the potential for iatrogenic lung injury. Mechanically resting lung volume is increased rather than decreased; however effective compliance may be reduced due to the higher lung volume which is caused by hyperinflation and air trapping.

CLINICAL ASSESSMENT OF DYNAMIC HYPERINFLATION

- *Direct auscultation:*
  Making sure expiration is complete before the next breath is delivered.
- *Measuring chest girth:*
  Place a measuring tape around the patient's chest at the level of the nipples. The increase in chest girth with each breath can be noted if dynamic hyperinflation is occurring.
- *End-expiratory occlusion test:*
  This test assesses the severity of dynamic hyperinflation.

- *Measurements of plateau pressure:*
  The plateau pressure will be higher than 30 cm H_2O. An attempt is made to keep the plateau pressure below 30 cm H_2O to avoid lung injury.

CLINICAL SIGNIFICANCE OF PULMONARY HYPERINFLATION

- *Impairing ventilation perfusion matching:*
  Over inflated regions may also compress more healthy regions of the lung impairing ventilation perfusion matching.
- *Inspiratory muscle overload:*
  These regions of overinflation put inspiratory muscles at substantial mechanical disadvantage which further worsens respiratory muscle function.
- *Load to trigger mechanical breath:*
  Regions of air trapping and intrinsic PEEP also function as a threshold load to trigger mechanical breath causing difficulty in weaning.

Monitoring

- *Peak flow rate:*
  PFR < 30% of the predicted normal signifies an asthmatic attack. Peak flow to be measured after initiating treatment and twice a day throughout hospital stay. Serial peak flow charting gives a good idea of improvement or worsening of acute attack.
- *Arterial blood gases:*
  Markers of severe attacks are a $PaO_2 < 60$ mmHg, a normal or rising $PaCO_2$ and respiratory or metabolic acidosis. Patients with acute severe asthma, high inspiratory overloading leads to respiratory muscle fatigue. The earliest sign of fatigue is normalisation of previously low $PaCO_2$ and rising $PaCO_2$ and acidosis is delayed sign.
- *Chest radiography:*
  The common finding is hyperinflated lungs. A consolidation or collapse secondary to mucous plugging or co-existing cardiac failure may also be detected.
- *Electrolytes:*
  A hypokalaemia is a common finding, which increases risk of cardiac arrhythmias. Patients on beta agonist and theophyllines are prone to have hypokalaemia.
- *Haematology:*
  A marker to infection which may lead to asthma attack is a leukocytosis.
- *Sputum analysis:*
  Purulent appearance of sputum is due to sputum eosinophilia. Hence a culture and Gram stain should be done to rule out bacterial infection.

INDICATIONS FOR INTUBATION AND VENTILATION

The decision to ventilate a patient is made on clinical grounds. The clinical trend is more important than the initial findings. Blood gas parameters are less useful in these patients. Evaluate the patient for clinical conditions in relation with changing trends of serial blood gases. Hypercapnia is not an indication for intubation because most episodes respond to bronchodilator therapy or noninvasive ventilation. Only 5% of asthmatics patient need intubation and mechanical ventilation. Decision for ventilating the patient depends on many factors:

- Cardiac or respiratory arrest
- Depressed level of consciousness with inability to protect airway
- Marked agitation
- Inability to cooperate with therapy
- Progressive respiratory muscle fatigue and exhausted patient
- Silent chest

  Wheezing and coarse airway crepitations may disappear as airway obstruction worsens. The silent lung being the sign of severe airway obstruction.
- Worsening hypoxia ($PaO_2 < 60$ mmHg) or cyanosis despite maximal oxygen concentration via face mask (FiO_2 of 60%)
- Hypercapnia—rising $PaCO_2$ with serial blood gas measurements
- Respiratory rate > 35 breaths/min
- Heart rate >120 beats/min.

ANAESTHETIC MANAGEMENT FOR VENTILATING PATIENT

Endotracheal intubation

Intubation should be smooth and by skilled personnel; repeated manipulation and failed intubation in patients with asthma may prove disastrous. Endotracheal intubation may be performed orally or nasally. Important advantage of oral intubation is it allows insertion of larger endotracheal tube which facilitates suctioning and offers less resistance. Many patients may experience get worsening of severe bronchospasm if a deeper plane is not maintained.

Drugs Used to Facilitate Intubation

Induction Agents

Intravenous induction agents of choice can be used which are not contraindicated in asthma.

- *Propofol:*

  Propofol is a better choice, which is a potent, fast acting sedative, decreases airway resistance after intubation compared to thiopentone. Propofol may cause hypotension.

- *Etiomidate:*

  Etiomidate is a better choice in haemodynamically unstable patients.
- *Ketamine*

  Ketamine does not cause hypotension or respiratory depression, however it increases laryngeal reflexes and predisposes to laryngospasm with excessive upper airway manipulation. It is used with caution.

Muscle Relaxants

Succinylcholine can produce histamine release but it is clinically insignificant, however it is advisable to avoid if possible. Any other nondepolarising neuromuscular blocking drug can be used for intubation except atracurium.

Sedative Drugs

Sedative drugs used to facilitate intubation reduce vascular tone and in combination with an induction agent decreases venous return, secondary to marked pulmonary hyperinflation. The decrease in venous return may contribute to marked haemodynamic instability at time of intubation.

Precaution after Intubation

Postintubation hypotension should be anticipated in severe asthmatics. This can be managed by rapid fluid administration and manual ventilation via Ambu bag at a slow rate to limit the severity of hyperinflation.

Strategy for Ventilation in Severe Asthmatics

In severe asthmatics, management can be easier if the ventilator strategy and pharmacological strategy is understood properly.

Ventilator Strategy

Though overall goals for ventilating severe asthmatics are similar like to other types of respiratory failure, to provide adequate oxygenation with nontoxic levels of inspired oxygen sufficient minute ventilation to achieve a normal arterial carbon dioxide tension and arterial pH. However, the primary focus of management of respiratory failure in patients with obstructive lung disease should be more towards improvement of deranged mechanics than to improve gas exchange.

1. *Initially provide substantial muscle unloading:*

   Ventilator muscle overload and fatigue are common manifestations of respiratory failure in obstructed patient. Therefore, selecting modes that initially provide substantial muscle unloading is particularly important.

2. *Maintenance of oxygenation is not a problem:*
   Avoid hypoxia by providing adequate gas exchange while minimising lung injury (Minimise peak airway and plateau pressure- $P_{plateau} \leq$ 30 cm H_2O). Although V/Q mismatch does occur in case of air flow obstruction, shunts are less of a problem and thus oxygenation usually can be maintained reasonably well without need to use toxic levels of inspired oxygen concentration.
3. *Optimisation of expiratory time:*
   Optimisation of expiratory time should take priority over the normalisation of blood gases. Because bronchospasm increases airway resistance several times above normal, time constants for exhalation are significantly prolonged in acute severe asthma.
4. *Early sedation and muscle relaxation:*
   An increasingly higher level of CO_2, in a dyspneic patient, from small tidal volume and/or air trapping, may require substantial amounts of sedation and sometimes muscle relaxation to have synchrony with ventilator.
5. *Controlled hypoventilation:*
   The biggest challenge is to provide physiologically adequate minute volume, while minimising risk of dynamic hyperinflation, auto-PEEP and barotrauma. In status asthmaticus attempts to correct respiratory acidosis, i.e. eucapnia by increasing minute ventilation may lead to extreme pulmonary hyperinflation. A rapid normalisation of the $PaCO_2$ leads to hypocapnic alkalosis. This can cause attacks of tetany, exaggerate bronchospasm, arrhythmias and electrolyte disturbances. The risk of tension pneumothorax and haemodynamic collapse may increase with pulmonary hyperinflation.

   The best objective to ventilate asthmatics is controlled hypoventilation with permissive hypercapnia. Rationale for this approach is that hypercapnia has got less risk than increased lung volumes. The high peak inspiratory pressure was avoided at any cost by setting a limit for peak inspiratory pressure 30 cmH_2O and is to be achieved by reducing tidal volume, respiratory rate, minute ventilation and inspiratory flows. This deliberate hypoventilation resulted in $PaCO_2$ level up to 60-70 mm Hg which were accepted and tolerated by most patients without complications.

HYPERCAPNIA

Patients can have hypercapnia despite normal or increased minute ventilation, which can only result either from an increase in CO_2

production or an increase in physiological dead space. It is presumed that hypercapnia which is a common finding during mechanically ventilated patients with status asthmaticus is caused by a marked increase in dead space that results from alveolar overdistension.

- In normal lung mechanics an increase in minute ventilation will lower $PaCO_2$ (doubling the rate should decrease $PaCO_2$ by 50%). However. this is not seen in patients with status asthmatics because, when minute ventilation is increased, the resulting increase in dynamic hyperinflation may further increase dead space.
- In status asthmatics, efforts to reduce an elevated $PaCO_2$ by increasing minute ventilation may be relatively ineffective and may not be able to normalise the $PaCO_2$ through ventilator manipulation.

Instead of correcting hypercapnia which gives rise to pulmonary hyperinflation, a reasonable approach is to choose ventilator settings that will provide safe and minimum level of dynamic hyperinflation. Set tidal volume 8-9 ml/kg, respiratory rate 12-14 breaths per minute and accept the resulting $PaCO_2$. Fortunately hypercapnia in status asthmatics seems to be generally well tolerated, rarely with serious consequences. Hypercapnia in status asthmatics is acute at the onset and therefore is not compensated by increased bicarbonate.

Treatment of Acute Hypercapnia in Status Asthmatics

Bicarbonate

One approach to acute hypercapnia is to ignore the elevated $PaCO_2$ and administer buffering agents to correct acidosis. Unlike metabolic acidosis however buffering acute respiratory acidosis with bicarbonate is relatively inefficient. Due to the loss of administered bicarbonate in the urine and generation of CO_2 through buffering agent, even partial correction of severe acidosis will require a minimum of several hundred milliequivalent of sodium bicarbonate in an adult.

- Such high doses will increase intracerebral blood flow and intracranial pressure. It is usually recommended to give a lesser amount of bicarbonate slowly, to only partially correct pH to 7.15-7.2 to minimise effect on ICP.
- Another problem with the use of large amounts of bicarbonate is that with resolution of airflow obstruction and hypercapnia, the patient is left with therapeutically induced metabolic alkalosis.

As a general rule, unless there is some compelling reason to correct underlying respiratory acidosis, e.g. arrhythmia, hyperkalaemia or unexplained haemodynamic instability, it is desirable not to correct serum pH. It is advisable to wait for the $PaCO_2$ to decrease as airflow obstruction improves.

If bicarbonate therapy is given, ideally it should be administered by slow infusion rather than by rapid bolus administration. Because the latter may lead to an acute increase in CO_2 production and a transient fall in intracellular pH as a consequence of rapid diffusion of CO_2 into cells. Fortunately many patients experience substantial improvement in their hypercapnia during the first 12 hours of intubation.

Tromethamine (THAM)

An alternative to sodium bicarbonate is tromethamine (THAM), a buffer that does not generate CO_2 during the buffering process. Even though THAM may offer some theoretical advantages over sodium bicarbonate for buffering acidosis, its use may lead to the same problem of posthypercapnic metabolic alkalosis.

INITIAL VENTILATOR SETTINGS FOR SEVERE AIRFLOW OBSTRUCTION—GENERAL GUIDELINES

- Selection of mode–Assist/Control
- FiO_2 - $SaO_2 > 90\%$
- Tidal volume–8-9 ml/kg (on volume ventilation)
- Respiratory rate–12-14 breaths/min
- Inspiratory flow as low as possible–60-80 liters/min (on volume ventilation)
- Inspiratory time (on pressure ventilation 0.8-1.2 sec)
- Waveform–Decelerating or Square
- Extrinsic PEEP–To offset 80-90% of auto–PEEP.

Selection of Mode

The choice of mode of ventilator depends on the clinical picture. Mostly ventilatory failure is due to respiratory muscle fatigue, the selected mode is to reduce the work of breathing and the resting of the respiratory musculature. Substantial muscle unloading generally is required in the initial management of patients with airflow obstruction and thus assist/control (A/C) is often indicated. VACV (volume assist/control ventilation) and PACV (pressure assist/control ventilation) are effective in this regard. Many clinicians prefer pressure ventilation over volume ventilation to allow the patient to set the peak inspiratory flow needed.

- *Volume assist/control:*

  If CO_2 clearance is believed to be more important than overdistension or synchrony, VACV should be the choice.

- *Pressure assist/control < 35 cm H_2O providing adequate volume:*
  If prevention of overdistension or synchrony is more important than CO_2 clearance, PACV should be the choice.
  - PACV offers very high initial flows (decelerating flow pattern that varies with the patient's efforts, and helps to keep the inspiratory time short and thus expiratory time long.
  - If patient is triggering breaths, since initial flow demands are high patient efforts are better synchronised than with other flow patterns.
  - Pressure limited breaths also impose pressure limit on the airway in spite of changing lung mechanics. If this is set at 35 cm H_2O or below, the risk of over distension injury is relatively less.

However, one should remember that PACV provides variable levels of minute ventilation depending on the patient's effort and lung mechanics and therefore do not offer guaranteed volume like VACV.

Inspired O_2 Concentration

Elimination of hypoxaemia can almost always be achieved by short-term use of high inspired O_2 concentration. FiO_2 adjustments generally can be maintained below 60% in these patients to deliver adequate oxygenation. FiO_2 should be kept between (30-50%). Maintain SaO_2 >90%. Lower values can impair respiratory function and higher values can suppress respiratory drive.

Minute Ventilation—Tidal Volume and Respiratory Rate

Minute ventilation is most the important determinant of dynamic hyperinflation leading to hypotension and barotrauma. This demonstrates there is a potential risk of progressively increasing minute ventilation in an attempt to correct hypercapnia.

In severe asthma during mechanical ventilation, three key factors determine the degree of volume trap:

- Resistance to airflow during expiration
- Tidal volume that must be exhaled
- Time for expiration

The key ventilator settings that influence the severity of hyperinflation are:

- Tidal volume
- Respiratory rate
- Inspiratory flow rate.

Low Tidal Volume – 8-9 ml/kg

Reducing tidal volumes as much as possible is therefore desirable, and decreasing pH and increasing partial pressure of carbon dioxide is a usually acceptable. When initiating controlled ventilation in asthmatic patients, tidal volumes may need to be reduced to avoid high airway pressure ($P_{peak} < 40$ cmH_2O). The tidal volume should be set as low as possible in accordance with CO_2 goals to keep maximal alveolar pressures ($P_{Plateau}$ pressure) at less than 30 cmH_2O. However, maximum plateau pressure can go upto 35 cmH_2O. The minute volume should be adjusted to pH 7.35 – 7.45 and not by $PaCO_2$ to avoid over ventilation with consequent alkali loss and reduced renal compensation. Respiratory acidosis seems to be well tolerated in patients without severe cardiac or CNS difficulties.

Low Respiratory Rate–8-9 Breaths/min

When there is a large increase in airway resistance, enough time has to be allowed for expiration to prevent an increase in air trapping. Increasing the respiratory rate leads to a significant increase in intrinsic PEEP as a result of reduced expiratory time. Low rates (6-8 breaths/min) are needed when the patient is alert and higher rates when the patient is sleeping to avoid spontaneous ventilation.

With respiratory frequency as low as 8 breaths/min. and I:E ratio 1:2, the total time for each breath is 7.5 seconds, with 5 seconds available for expiration. A further decrease to 6 breaths per minute can allow 6.7 seconds for expiration which is sufficient for most of the severe cases. I: E ratio 1:3 or 1:4 will reduce inspiratory time and also inspiratory pause. Inspiratory pause is useful for mean alveolar/mean airway pressure, which is responsible for efficiency of CO_2 removal and oxygenation. Extending the I: E ratio to 1:3 may be necessary in some cases to avoid progressive air trapping.

Inspiratory Flow Rate (Volume Controlled Ventilation)

Use of decelerating flow pattern is recommended and the flow rate is kept according to breathing pattern. High flows are recommended to satisfy inspiratory demands, to shorten inspiratory time and to prevent development of auto-PEEP. Patients with a high level of respiratory drive and slow expiratory flow (such as in severe airway obstruction) needs rapid inspiratory flow rates in mechanical ventilation. If the inspiratory flow rate is lower than a patient's demand, additional inspiratory workload will be imposed on the patient.

High inspiratory flow rates (> 60-80 lit/min) will satisfy most patients inspiratory flow rate demand thereby reducing intrinsic PEEP, dynamic hyperinflation and physiological dead space.

Prolongation of Expiratory Time

Effects of prolongation of expiratory time on dynamic hyperinflation can be assessed by $P_{plateau}$ and auto-PEEP. As expected $P_{plateau}$ and auto-PEEP decreases when expiratory time is prolonged but the magnitude of this reduction was not profound.

Total pulmonary hyperinflation has two components:

- Dynamic component that is amenable to ventilatory manipulation
- The second component is caused by trapped gas behind occluded airways (baby lung) which is therefore not influenced by prolongation of expiratory time.

$P_{plateau}$ and Auto-PEEP

Very high respiratory rates and very short expiratory times should be avoided. There is often little to be gained by reducing the respiratory rate below 12 -14 breaths per minute when tidal volume of 8-9 ml/kg is used. An exception might be when hyperinflation is marked ($P_{plateau}$ >30 cm H_2O or has resulted in complications) then even a small reduction in hyperinflation could have a clinical impact.

$P_{plateau}$ is not affected by inspiratory flow-resistive properties and may be a better parameter for monitoring lung hyperinflation. At constant tidal volume, changes in the degree of dynamic hyperinflation in response to bronchodilators or changes in expiratory time can be determined from changes in plateau pressure. Because $P_{plateau}$ represents end inspiratory elastic recoil pressure and provides an estimate of average peak alveolar pressure, it also might help to predict a risk of alveolar rupture. Although the threshold plateau that increased risk of barotrauma is not well defined in status asthmaticus an acceptable upper limit of 25-30 cm H_2O has been suggested.

Inspiratory Time (On Pressure Ventilation 0.8-1.2 Sec)

It should be adequate for comfort and avoidance of unnecessary high peak (i.e. flow resistive) pressures (e.g. 0.8-1.2 sec). However, when setting inspiratory time, clinicians must take into account frequency (and thus I/E ratio) as they attempt to balance required alveolar ventilation with the risk of air trapping.

Role of Extrinsic PEEP—How Much to Start PEEP?

The impact of extrinsic PEEP on lung volume is different in COPD and asthma. Extrinsic PEEP does not affect lung volume or airway pressure in COPD but both were increased in asthma. A patient receiving controlled ventilation with deep sedation or paralysis minimal PEEP ≤ 5 cm H_2O levels should be used.

A patient with status asthmaticus breathes at a very high resting lung volume, fighting to decrease airway obstruction by increasing outward stretch of the intrapulmonary bronchi. In asthmatics the communicating gas volume is low because most the air is trapped behind closed airways. PEEP can be increased slowly being aware of circulatory complication (depression of cardiac output). Only application of PEEP can break the life threatening bronchospasm when pharmacological bronchodilators fail.

PEEP open up closed spastic airways during inspiration and keep them open during expiration. Auscultation of the chest reveals a silent chest followed by audible bronchospasm and finally closes to normal lung sounds, when PEEP levels are increased. In spite of nebuliser therapy, if the chest remains silent increase set PEEP by 2 cm H_2O. Peak airway pressure usually drops and expired tidal volume or expired minute ventilation improves if bronchi start dilating.

Difficulty in Synchronisation

In patients with airflow obstruction the presence of air trapping and high levels of muscle loading can markedly increase the sense of dyspnoea and make it difficult to properly synchronise ventilator breath triggering, flow delivery and breath cycling with partial demand. Careful adjustments of inspiratory pressure, volume and timing therefore must be done to ensure proper assisted breath synchrony. Indeed, in some cases, judicious use of sedation to blunt the ventilatory drive may be necessary to achieve these goals. With assisted ventilation the intrinsic PEEP level is important for setting the trigger pressure at the ventilator.

The pressure P needed to trigger an ASB breath is given by the equation:

$$P = PEEP_i\text{-}PEEP_e$$

Through the application of $PEEP_e$, the trigger threshold for activating the next inspiration is reduced, so that the respiratory effort required for triggering is also reduced.

Monitoring

- Monitor expired V_T or minute ventilation
- Monitor P_{peak} airway pressure and $P_{peak} - P_{pause}$

Weaning

Asthma patients do not require ventilatory support for prolonged time. Settling of bronchospasm means the asthma attack is resolved in clinical ground, weaning can be attempted. Most can be successfully and easily weaned. Difficulty in weaning may be in patients with respiratory muscle weakness from hypokalaemia, hypophosphaetemia, excessive use of steroids and long term use of muscle relaxants.

PHARMACOLOGICAL STRATEGY IN SEVERE ASTHMA

- Adequate hydration
- Use bronchodilators, glucocorticoids
- Adequate sedation

Adequate Hydration

Acute asthmatic patients are mostly dehydrated especially in acute attacks due to reduced oral intake and excessive sweating. Adequate hydration with fluid can be given orally or intravenously. Oxygen must be well humidified. Mucolytics and expectorants should be avoided in acute attacks since their efficacy is unproven. Therapeutic bronchial lavage via bronchoscope to remove thick mucous plugs on ventilated patients is not routinely recommended since it produces significant desaturation and other complications.

Use Bronchodilators and Glucocorticoids

No specific studies of the use of bronchodilators in severe asthmatics during mechanical ventilation are available the way it is available for COPD. Since in severe asthma no specific studies are available, optimal number of nebulizations and dosing interval is determined by assessment of lung mechanics during incremental dosing of inhaled β_2 agonists.

Types of Beta Agonist

- *Nebulized β_2 agonist:*

  Nebulized β_2 agonists are the main stay of acute severe asthma. Hyperinflated asthmatics are too breathless to inhale a metered dose. Compressor driven nebulizer are recommended in these patients since delivery does not depend on coordination of breathing pattern and a large total dose can be given.

Nebulized β_2 agonist has to be diluted with 2 ml normal saline. In patients with severe bronchospasm nebulized drugs may have to be given initially at 15 minutes interval (maximum 3 times) depending on the extent of airway obstruction. If the spasm still does not improve, mechanical ventilation has to be considered. The frequency of nebulization ranges from 2-8 hours depending on the severity of the spasm. Nebulized salbutamol or terbutaline of 5 mg diluted with 2-3 ml of normal saline must be given quickly. The dose can be given every hourly (watch for toxicity, e.g. tachycardia) and then every 2-3 hourly if the patient shows good response. The patient must receive simultaneous oxygen because nebulizer driven air can worsen hypoxaemia due to improvement of ventilation. Hence, nebulization must be driven by oxygen instead of air or patient simultaneously should receive supplemental nasal oxygen. If inhalation of nebulized drugs are not effective than intravenous beta$_2$ agonist should be given.

- *Intravenous β_2 agonist:*
  Nebulized medication may not be able to reach affected airways due to extensive small airway mucous plugging which is seen in severe asthmatics. Rapid onset of action and high rate of toxicity (tremors, tachycardia) is also seen in these patients.
  Intravenous β_2 agonist (Terbutaline) 10 mg/min following bolus of 0.5 mg or salbutamol, 10 mg/min can be given as infusion.
- *Aerosols Involve in-line circuit nebulizer:*
  Aerosols involve in-line circuit nebulizer, although metered dose inhalers with inspiratory circuit holding chambers are also effective. Because endotracheal tubes significantly reduce aerosol delivery, doses are usually increased three to fourfold (or even aerosolized continuously) to ensure adequate drug effectiveness.

Ipratroprium Bromide

Nebulized ipratroprium bromide is not as potent a bronchodilator as salbutamol but the advantage is that it does not cause any tachycardia. The combination of salbutamol and ipratroprium bromide has an additive bronchodilator effect. Nebulized ipratroprium bromide can be given in a dose of 500 µg every 6 hours.

Aminophylline

Patients who have failed to respond nebulized beta-agonist and steroids alone, intravenous aminophylline is the drug of choice, especially in severely ill ICU admitted patients. If a Patient is not on oral theophyllines at the time of admission, initial loading dose is 6 mg/kg body weight diluted in 20 CC of 5% dextrose, – IV slowly over 20-30 minutes. The loading dose is increased in smokers (× 1.5), and reduced in patients with pneumonia or cardiac failure (× 0.4) and those with severe hypoxia (× 0.8). The loading dose is omitted in any form of oral theophyllines. Following the loading dose an infusion is set up at a rate of 0.6 to 0.9 mg/kg/hr. A maintenance drip IV – 2 amp of aminophylline in 500 ml of 5% dextrose – 10-14 drops per minute (0.5 mg/kg/hr). The plasma concentration of theophyllines should be maintained within the therapeutic range of 10-20 µg/ml. The adverse effects increases if serum levels are above 25 µg/ml. These adverse effects include nausea, headache, insomnia, hypotension, cardiac arrhythmias, convulsion and death. Liver disease, cardiac failure, pneumonia, hypoxemia and drugs, e.g. Macrolides and most quinolones affect theophylline clearance.

Steroids

Asthma is an airway inflammatory disease, steroids play a major role in acute severe asthma. Acute asthmatics admitted in the ICU should promptly receive systemic steroid therapy. Though there is no conclusive study regarding optimal dose or benefit of any particular type of steroid, the following doses are recommended.

Glucocorticoids are an essential component of the treatment of severe asthma and should be administered early, because effect is not seen in the first 6 hours; and mostly the effect is seen within 6-12 hours of administration.

- *Oral therapy:*
  In a patient not having severe asthmatic attack and no vomiting oral prednisolone in a dose of 40-60 mg can be given and the repeated daily. The oral therapy is as effective as intravenous steroids.
- *Intravenous therapy:*
  In critically ill asthmatics admitted in the ICU, intravenous hydrocortisone 200 mg stat is given and 100 mg is repeated every 6-8 hours (2-3 mg/kg).

Methyl prednisolone 125 mg IV stat – 40-80 mg every 6-8 hours can be given. Initial dose is 1-2 mg/kg per day of methylprednisolosne or equivalents are appropriate. Complications following short duration (7-14 days) and moderately high dose course of steroids are minimal. There is no proven study that giving a larger dose significantly speeds up and improves the response, however an initial mega dose precipitates acute steroid myopathy.

Sedation

Deep sedation and sometimes neuromuscular blockade is necessary.

Other Drugs for Severe Spasm

Adrenaline

Adrenaline is now less frequently used; instead nebulized beta agonists can be administered. Subcutaneous adrenaline is potent vasodilator. In patients with hypertension, elderly patients, pre-existing ischaemic disease adrenaline is potentially cardiotoxic. The usual dose is 0.5 ml of 1:1000 solution given subcutaneously. The maximum of 2 ml can be given if the patient is monitored in the ICU. Adrenaline 6 mg in 100 ml, 1-5 ml/hr infusion can be also given, after careful monitoring haemodynamic status.

Antibiotics

Routine administration of antibiotics is not recommended. Though infection is a common trigger, such infections are usually viral. Antibiotics if bacterial infection is diagnosed or suspected.

Augmentin – (amoxicillin + clavulanic acid)–Amoxyclav – 1.2 gms IV 8 hourly with Macrolides – IV azythromycin 500 mg OD or clarythromycin 500 mg BD

Or quinolones – levofloxacin 500 mg IV OD

Or third generation cephalosporin

Cefotaxime + sulbactum – 1.5 gm 8 hourly

Assessment of airway pressures (peak to plateau gradients) or flow-volume pattern can be used to monitor bronchodilator effectiveness.

Magnesium Sulfate

$MgSO_4$ is a safe and cheap form of therapy, that can be considered in all refractory cases. In adults pulmonary function improves if 2 gm of $MgSO_4$ given as a 20 minute IV infusion, when used as an adjunct to standard therapy. Recent studies have recommended that isotonic solution of nebulized $MgSO_4$ was a useful adjuvant to salbutamol in the treatment of severe asthma.

Sedation in ICU

Sedatives are contraindicated in patients without ventilatory support. The goal to sedate mechanically ventilated patient in ICU includes provision of amnesia, anxiolysis and analgesia and prevention of patient ventilator dyssynchrony. In status asthmatics sedatives have got an additional role because controlled hypoventilation in spite of acute respiratory acidosis increases respiratory drive.

A combination of propofol or a benzodiazepine (midazolam or lorazepam) with narcotics (fentanyl or morphine or buprenorphine) often proves optimal and very large doses may be required. Since both propofol and midazolam have similar properties use any one.

- *Propofol*
  1.2 to 1.5 mg/kg bolus 0.8 mg/kg/hr–Propofol
- *Midazolam*
  Infusion 1mg/ml

Advantage of propofol:

One advantage of propofol over benzodiazepines in severe asthma is that this agent often allows deep sedation but reverses promptly on its discontinuation.

A maximal dose of approximately 5 mg/kg per hour is recommended because prolonged infusion of very high doses of propofol can lead to life–threatening complications.

Ketamine – not good choice:

Ketamine an intravenous dissociative anaesthetic has also been used in asthma. This drug however can lead to significant increases in blood pressure, heart rate, and intracranial pressure. Their seems little justification for the use of ketamine in ventilated patient with status asthmatics.

Precautions:

Because most patients improve significantly within 24-48 hours prolonged residual sedation that delays extubation is undesirable. Large doses of sedatives and narcotics in combination with marked lung inflation may lead to hypotension, secondary to decreased venous return. When additional muscle relaxation is needed, for better haemodynamic stability, it may be safer to administer a nondepolarizing neuromuscular blocking agent (NMBA) than to increase the dose of sedatives or narcotics.

Neuromuscular Blocking Drugs

In extreme cases, it may be necessary to paralyse patients to prevent "fighting" with the ventilator and generation of high airway pressures.

Mode of administration of neuromuscular blocking agents (NMBA):

The best way to administer drugs in asthmatics are intermittent. Even when hypotension is not present, intermittent administration of one or more boluses of a NMBA may help to provide temporary muscle relaxation, during which time sedative doses can be increased or decreased gradually to reach target level. Prolonged use of an NMBA may increase the likelihood of myopathy in status asthmatics, but short term use does not carry significant risk. The specific NMBA is not important, but avoid pancuronium in renal failure and atracurium in asthma.

Intermittent bolusing is preferred to a continuous infusion because this permits continuous assessment of the adequacy of sedation and lessens the likelihood that the patient will undergo unnecessarily prolonged neuromuscular paralysis. When sedatives and narcotics are used liberally along with short term intermittent boluses of a NMBA as per requirement, very few patients with status asthmatics will require prolonged continuous neuromuscular paralysis.

Complications in Ventilating Severe Asthmatics

Common complications are nosocomial pneumonia, sinusitis, pulmonary embolism and gastrointestinal bleeding. Additional complications that result from severe asthma itself or from medications used (to treat airflow obstruction, to provide muscle relaxation). These include hypotension, barotrauma, myocardial injury, rhabdomyolysis, lactic acidosis, neurologic injury and acute myopathy.

Complications due to Dynamic Hyperinflation

The two major complications of barotrauma and circulatory depression with hypotension are attributed to dynamic hyperinflation.

Hypotension

In status asthmaticus the most common cause of hypotension is excessive pulmonary hyperinflation that impedes venous return. Extreme hyperinflation can also lead to cardiac arrest. Sedatives producing myocardial depression and decreasing venous return or pneumothorax can also give rise to hypotension. If the one minute apnoea trial and a rapid infusion of fluid do not restore blood pressure, then hypotension may be due to pneumothorax.

High intrathoracic pressure impairs venous return and results in a decrease in cardiac output. The drug which increases venous capacitance such as morphine or diazepam or hypovolaemia exaggerates fall in cardiac output. The effect can be minimised by maintaining adequate intravascular volume and shortening inspiratory phase of ventilation.

Hypotension in a mechanical ventilated patient with severe asthma always rules out dynamic hyperinflation. A brief trial of apnea (30-45 seconds) helps to differentiate if hypotension is due to dynamic hyperinflation. During apnea period venous return increases which raises blood pressure. If hypotension is due to dynamic hyperinflation, increase fluid administration and decrease respiratory rate. If hypotension does not respond to fluid challenge, then most common possibilities are tension pneumothorax or myocardial depression.

Pneumothorax

Barotrauma includes interstitial emphysema, pneumomediastinum. pneumoperitoneum, subcutaneous emphysema, and pneumothorax. Pneumothorax is not only common in status asthmaticus due to pulmonary hyperinflation but also dangerous. The hyperinflated lungs resist collapse and allow small pneumothorax to be under tension. resulting in rapid deterioration and sometimes death. Injury to hyperinflated lungs should be avoided by placing chest tubes by blunt dissection than blind trocar method.

Cardiac Complications

The stunned myocardium of status asthmaticus is secondary to massive catecholamine release. This is also seen after severe psychological stress or any conditions associated with massive endogenous sympathetic activation and typically have a benign course. Patients may have decreased myocardial contractility and deep T wave inversion in the electrocardiogram, simulating myocardial ischemia. During an acute attack of hypoxia, or drugs used in asthma such as theophyllines, beta agonist can give rise to arrhythmias.

Hypokalaemia

Asthma induced respiratory alkalosis and from steroids and beta agonist used in the treatment of asthma can give rise to hypokalaemia.

Rhabdomyolysis

Rhabdomyolysis is noted with patient received prolonged infusion of propofol in very high doses or as a result of extreme muscular exertion coupled with hypoxia.

Lactic acidosis

Lactic acidosis is relatively common in status asthmatics. Even a moderate degree of lactic acidosis is dangerous in patients with significant hypercapnia. Lactic acidosis is attributed to lactate production by respiratory muscles. This is more common when beta 2 agonists are given intravenously or in excessively high doses of inhalational therapy.

Central Nervous System Injury

Hypercapnia poses little risk for long term neurologic sequelae. Hypercapnia induced increase in intracranial pressure potentially could worsen brain injury following cardio-respiratory arrest. Cerebral anoxia is the most common cause of death in patients with status asthmatics. Rarely cerebral oedema or subarachnoid haemorrhage in status asthmatics that occurred in the absence of prior cerebral anoxia have been attributed to hypercapnia.

Myopathy CCPM—Critical Care Polyneuropathy

Acute myopathy probably is the most common cause of morbidity affecting patients with asthma who undergo mechanical ventilation. The pathogenesis is incompletely understood, but it usually has been attributed to the combined effects of glucocorticoids and prolonged neuromuscular paralysis or deep sedation.

Myopathy, however can also occur with glucocorticoids and prolonged ICU stay >5-7 days, mechanical ventilation under deep sedation without paralysis. It is possible that prolonged near total muscle inactivity, whether induced by neuromuscular paralysis or by deep sedation, may increase the sensitivity of muscles to the myotoxic effects of corticosteroids.

Beta Agonist

These drugs given intravenously or through nebulization can give rise tremors, hypokalaemia and arrhythmias.

Steroids

Acute gastrointestinal bleeding and hypokalaemia can be seen after the use of steroids.

SUMMARY

Settings	*Precautions to be taken to avoid*
Low tidal volume	Dynamic hyperinflation
Low respiratory rate	Hypotension
Low minute ventilation (permissive hypercapnia)	High airway pressures
Prolonged expiratory time	Pneumothorax

CHAPTER

39

Ventilatory Management of Chronic Obstructive Pulmonary Diseases

COPD is defined as airway limitation, which is usually non-reversible and associated with

- Hypertrophy and hyperplasia of mucous glands
- Increase in number of mucous secreting cells, e.g. goblet cells
- Widening of air spaces distal to the terminal bronchiole
- Destruction of lung parenchyma.

PRECIPITATING FACTORS

There are various precipitating factors which are responsible for acute respiratory failure in patients with chronic airway obstruction:-

- *Acute infection:*
  The most important precipitating factor is acute infection causing an exacerbation of bronchitis giving rise to bronchoconstriction, mucosal oedema, inflammation and increased secretions.
- *Use of sedatives and respiratory depressant drugs:*
  This is mostly seen in postoperative patients and due to a variety of reasons. Suppression of cough due to pain in critically ill patients leads to sputum retention and worsening of respiratory failure. Depressant effects of anaesthetic, sedative, histamine release by drugs such as succinylcholine, morphine can be dangerous in these patients.
- *Environmental factors:*
  Polluted air also precipitates acute crisis.

PATHOGENESIS

In exacerbation of COPD, the fundamental feature is inefficient gas exchange giving rise to respiratory failure (Ventilatory pump failure). A worsening in COPD consists of:

- Increase in ventilation perfusion mismatch leading to hypoxaemia.
- Fall in alveolar ventilation with a rise of carbon dioxide in the blood. This rise is the effect of decrease in minute ventilation and/or increase in dead space.

If ventilation perfusion mismatch is the only abnormality the patient will be having only hypoxaemia. If the patient is having ventilation perfusion mismatch, associated with a decrease in alveolar ventilation he will be having hypoxaemia and hypercapnia.

- *Ventilation perfusion mismatch:*
  Ventilation perfusion mismatch is present in stable COPD patients as a result of parenchymal destruction and small airway disease. During exacerbation of COPD, further impairment of gas exchange is due to worsening of ventilation perfusion mismatching, and increased peripheral oxygen uptake is due to increased effort of inspiratory muscles which are overloaded.
- *Increase in dead space:*
  Underlying emphysematous changes results in capillary loss and increasing of dead space with over distended regions of the lung, thus increasing dead space ventilation. This wasted ventilation further compromises the ability of the respiratory muscles to supply adequate ventilation for alveolar gas exchange. Hypoxaemic pulmonary vasoconstriction associated with chronic pulmonary vascular changes in some airway diseases, overload the right ventricle, further decreasing blood flow to the lungs and making the dead space larger.
- *Shunt:*
  Although V/Q mismatch does occur in case of COPD, shunts are less of a problem and thus usually oxygenation can be maintained reasonably well without having to use toxic levels of inspired oxygen concentration (FiO_2). Administration of oxygen rich air is effective in keeping oxyhaemoglobin saturation greater than 90%.

DIAGNOSIS

The feature of an acute exacerbation of COPD is increasing dyspnea, purulent sputum, increasing quantity of sputum. Sign of acute airway obstruction such as use of accessory muscle of respiration, rhonchi, prolonged expiration and an over inflated chest often is observed. Flapping tremors are present with mild to moderate rise in $PaCO_2$ but may be absent with a marked rise in $PaCO_2$. Warm peripheries are not always observed with hypercapnia. A patient with cyanosis, coma, drowsiness and apathy denotes a grave situation. Carbon dioxide retention is an important cause of metabolic coma.

IMPORTANT CLINICAL MANIFESTATION IN EXACERBATION OF COPD

Hypercapnia

Exacerbation of COPD can lead to hypercapnia [arterial carbon dioxide tension ($PaCO_2$) is > 45 mmHg], when breathing room air. This is due to a high respiratory rate and low tidal volumes, leading to decreased alveolar ventilation. Hypoxaemia is mostly associated with hypercapnia.

Respiratory Acidosis (pH < 7.36)

Respiratory acidosis represents a $PaCO_2$ that is higher than normal (> 45 mmHg), leading to arterial blood pH less than 7.36. When the lungs cannot remove all the CO_2 produced by the body, or when alveolar ventilation is decreased, respiratory acidosis occurs. One important reason for hypercapnia is inappropriately increased supplemental oxygen, which blunts hypoxic drive of ventilation.

Correction of respiratory acidosis should be performed carefully. The goal is to return the pH towards normal limits, not to return $PaCO_2$ to normal. Vigorous increase in minute ventilation should be avoided because of the risk of dynamic hyperinflation. If intracranial hypertension and overt haemodynamic instability do not exist but dynamic hyperinflation is a concern, acceptance of acidaemia (pH > 7.2) may be reasonable.

Intrinsic PEEP

The degree of intrinsic PEEP is related to the severity of resting hypercapnia. Exacerbation of COPD is mainly due to increased inflammation and altered mucous transport, particularly in small airways, which increases airway resistance. Severe airflow obstruction (due to mucosal oedema, spasm and secretion) results in air trapping, a reduced chest wall compliance and dynamic hyperinflation.

This significant increase in airway resistance gives rise to:

- Prolongation of inspiratory time
- Inadequate time for expiration
- Augmented inspiratory effort to ventilate the lungs
- Retarded rate of lung emptying.

As a consequence, lungs cannot properly empty and return to their normal "resting volume". At higher end-expiratory volumes (air trapping), an elastic force generates a positive pressure in the alveolus.

This elastic recoil pressure is manifested as intrinsic positive end-expiratory alveolar pressure ($PEEP_i$ or auto-PEEP). This insufficient decompression gives rise to dynamic pulmonary hyperinflation. This increases workload on the inspiratory muscles to generate negative intrapleural pressure required to initiate inspiratory airflow.

The consequent of air trapping is inspiratory muscles increases its lengths; however their optimal length tension relationship is exceeded. They therefore work at a mechanical disadvantage. Compared to normal subject's the diaphragm is less efficient to assist in inspiration. These combinations of factors of increased work of breathing, reduced mechanical efficiency and capability of respiratory muscle, together with dynamic hyperinflation can lead to progressive respiratory muscle fatigue, increasing alveolar hypoventilation and even respiratory arrest.

Stable COPD patients have few cm H_2O of intrinsic PEEP but it increases significantly during acute exacerbation. Patients ventilated for acute severe asthma or exacerbations of chronic obstructive lung disease have measurable levels of auto-PEEP which may be as high as 15-20 cm H_2O. Intrinsic PEEP must be first counterbalanced by the contracting inspiratory muscles in order to produce inspiratory flow. This inspiratory threshold load produces increased work of breathing and challenges the ability of the respiratory muscle to clear carbon dioxide. The degree of intrinsic PEEP is always related to the severity of resting hypercapnia. Compensatory mechanisms exist but are inadequate.

The combination of increased muscle load and reduced respiratory muscle force may lead to respiratory muscle fatigue and eventual exhaustion of the ventilatory pump. Assisted ventilation is needed at this juncture which will save the life of the patient.

The effects of intrinsic PEEP in COPD depend on the mechanism of intrinsic PEEP and its magnitude. In some patients the level of intrinsic PEEP is so high that without the addition of PEEP the patient cannot trigger the ventilator. The resulting ineffective efforts unduly increase the respiratory workload. Intrinsic PEEP is caused by both intrapulmonary and extrapulmonary factors.

Factors Determining Dynamic Hyperinflation and Intrinsic PEEP

Patient's respiratory mechanics:

Given the long time constants in obstructive air disease (resulting from high resistance and often high compliance units), the risk of air trapping is high. The clinical challenge is thus to reduce the total ventilation and I:E ratio as much as possible to minimise the risk.

Added flow resistance
- Endotracheal tube
- Ventilator tubings and circuit

Patient breathing pattern and ventilatory settings
- Tidal volume
- Frequency
- T_i/T_{tot}
- End-inspiratory pause

Indications for Ventilation

Mechanical ventilation should be considered when hypercapnia and respiratory acidosis, which is associated with breathlessness and tachypnoea, persist or worsen despite optimal and aggressive medical therapy.

- To alleviate distressing breathlessness
- Low respiratory frequency due to extreme hyperinflation-Arterial blood gas must be done to confirm that dyspnoea is related to respiratory acidosis.

The main characteristics of ventilator dependent patients with extreme COPD are:

- Shallow breathing pattern
- High airway occlusion pressure $P_{0.1}$, which is an index of overall neuromuscular drive
- High effective inspiratory impedance ratio:

$$P_{0.1}/V_T/T_I \text{ ratio}$$

Tidal volume V_T/Inspiratory time T_I = Mean inspiratory flow

A ratio greater than 10 cm H_2O/liter/second denotes inability to sustain spontaneous breathing for more than a few minutes. The ineffective compensatory mechanism with the low V_T causes chronic CO_2 retention. These patients with COPD develop a high respiratory drive to cope with excessive mechanical load.

STRATEGY FOR CHRONIC AIRFLOW LIMITATION

The management strategy can be divided into ventilator strategy and pharmacological strategy.

Ventilator Strategy

The primary focus of management of patients with respiratory failure due to obstructive lung disease should be more towards improvement of deranged mechanics than to improve gas exchange.

- To improve ventilation, correct V/Q abnormalities as far as possible, increase the PaO_2, correct a lowered pH and reduce the $PaCO_2$.
- Recognise and treat complications encountered during acute respiratory failure.

VENTILATOR ASSISTANCE

Ventilator assistance with exacerbation of COPD can be given in two ways:

1. **Non-invasive mechanical ventilation (NIMV)**
   - Non-invasive positive pressure ventilation (NPPV)
   - Negative pressure ventilation–iron lung.
2. **Traditional or conventional invasive mechanical ventilation via endotracheal tube**

1. Non-invasive Positive Pressure Ventilation (NPPV)

Patients with hypercapnia and respiratory acidosis, associated with breathlessness and tachypnoea and if they are able to cooperate with the intensivist and interact with the ventilator, first line of treatment in them is NPPV. This should be supplemented with optimal medical therapy and oxygen supplementation. Factors associated with the success of NPPV include a younger age, ability to cooperate, lower acuity of illness, availability of resources. The indication of NPPV in acute crisis in COPD is:

- Respiratory rate >30/min.
- Respiratory distress due to moderate or severe dyspnoea.
- pH ≤ 7.30 and/or a PaO_2 60 mmHg, provided these values fail to improve or worsen after trial of 6-8 hours (or even earlier) of conservative measures.

In the first few hours, NPPV requires the same level of settings as conventional ventilation and also is as effective as conventional ventilation in reversing acute respiratory failure secondary to decompensation of COPD. Adjustments of the mask and ventilator settings are essential during the first 30-60 min of NPPV; patients who show improvement in gas exchange within the first hour are more likely to avoid intubation.

Avoid NPPV in COPD if

- Obtunded patient, breaths poorly or irregularly
- Unable to protect airway
- Copious respiratory secretion
- Risk of aspirating gastric contents
- pH < 7.20, acute rise of $PaCO_2 > 70$ mmHg.
- Patient worsens on NPPV or fails to show improvement

NPPV decreases mortality from acute respiratory failure, need for endotracheal intubation in more than 50% patients when used as initial treatment. The use of NPPV was associated with a lesser risk of nosocomial infection, less antibiotic use and lower mortality. A face mask achieves more rapid correction of hypercapnia than a nasal mask during NPPV.

NPPV is successful when optimal medical therapy and oxygenation fail to reverse respiratory failure. Partial ventilatory support can be supplied successfully to patients with acute airflow obstruction using a noninvasive mask system. Generally these systems use pressure-targeted breaths, and they can be used either to prevent intubation in acute disease or to facilitate postextubation management. Though a variety of ventilatory modes can be used in NIPPV, BiPAP and PSV ventilation is most commonly used. Other used modes include pressure assist (patient-triggered, pressure targeted, time-cycled breaths). PEEP and CPAP formerly banned are now used commonly to counter balance PEEPi. When CPAP is used in spontaneous patients CPAP will reduce the work of breathing but dynamic hyperinflation is not a major risk.

- *BiPAP*

  BiPAP allows for good gas exchange and effectively reduces both the work of breathing and patient distress. Inspiratory pressure is kept around at 8 to 10 cm H_2O above the end-expiratory pressure. If end-expiratory pressure is 5 to 8 cm H_2O, the inspiratory pressure will be 13 to 16 cm H_2O. The pressure can be adjusted for effective alveolar ventilation and good exchange.

- *Pressure Support Ventilation/Assisted Spontaneous Breathing*

  Optimal PSV by face mask decreases respiratory muscle activity and workload in COPD patients. PSV does not worsen dynamic hyperinflation and intrinsic PEEP in some patients with COPD. PSV in COPD patient helps:

  - To reduce respiratory rate
  - To reduced intrinsic PEEP

- To reduced work of breathing
- Decreases O_2 demand and cost of breathing
- Reduces inspiratory time by manipulating inspiratory flow.

Ventilatory settings of PSV:

The most effective mode of NPPV is the combination of PSV 10-15 cm H_2O with CPAP 4-8 cm H_2O. Same level of pressure assistance, achieved better physiological results, if the portion of pressure is divided between CPAP and PSV rather than when delivered as PSV alone. This confirms the important role of the inspiratory workload in exacerbation of COPD patient. NIPPV does not improve ventilation perfusion mismatch significantly, but it increases alveolar ventilation by correcting the breathing pattern, which achieves more efficient clearance of arterial CO_2.

When to intubate patient on NPPV:

NPPV can be considered successful when ABGs and pH improve, dyspnea is relieved, the exacerbation resolves without intubation. In patients receiving NIPPV, intubation should be considered when NIPPV seems to fail, as shown by worsening of ABG over 1-2 hours or lack of improvement in ABG after 4 hours. Patients who show a sharp progressive rise in the $PaCO_2$ with a pH < 7.2 on controlled oxygen therapy, if dangerous hypoxia is unrelieved on conservative therapy or NPPV. Inpatients showing poor response to antibiotics, bronchodilator, and physiotherapy should be intubated and ventilated.

Endotracheal intubation and controlled ventilation offers two advantages:

- It allows proper access for suctioning of respiratory secretions
- It allows mechanical ventilatory support.

2. Traditional or Conventional Invasive Mechanical Ventilation via Endotracheal Tube in COPD Patient

The goal to remember is airway and alveolar pressures must be kept lower by allowing permissive hypercapnia. Although a plateau pressure greater than 30 cm H_2O is a useful marker for risk of lung stretch injury, clinicians must also be aware that high peak airway pressures, even in the presence of acceptable plateau pressures, may transiently overdistend the lung producing distension injury, because of pendelluft effect.

- *Controlled Mechanical Ventilation*

  CMV is less commonly used in the ICU than in the past because of wider use of NPPV and improved pharmacologic treatment. CMV can be used to measure passive respiratory mechanics.

- *End expiration occlusion test (EEO):*
  The rapid airway occlusion technique is for assessing respiratory mechanics based on airway opening pressure and flow measurements. When applied at the end of expiration (end expiration occlusion EEO), which measures intrinsic PEEP (auto-PEEP).
- *End inspiratory occlusion test (EIO):*
  When applied just before at the end of inspiration (end inspiratory occlusion EIO) it measures total respiratory resistance.
  During CMV dynamic intrinsic PEEP has to be counterbalanced by the positive pressure of the ventilator to start the inspiratory flow.

- *Assist Volume Control*

  Assist-volume control (AVC) ventilation was used in decompensated patients. In patients with COPD and high levels of intrinsic PEEP, high inspiratory flow rates are recommended (provided plateau pressure is not extended beyond 30 cm H_2O). High inspiratory flow rates shorten inspiratory time and increase expiratory time potentially reducing intrinsic PEEP. Tidal volume of about 7-9 ml/kg is prudent in COPD.
  The presence of narrowed airways can result in very high peak airway pressures to deliver normal tidal volume. Moreover, the development of intrinsic PEEP can further increase the inflation pressures delivered by a mechanical ventilator. These pressures, in setting of heterogeneous regional airflow characteristics, can result in transient overinflation of less obstructed regions by a pendelluft effect.

- *Assist Pressure Control*

  Assist pressure control (APC) ventilation, maximal airway and alveolar pressures are controlled. With rapid variations in patient impedance, as in acute COPD, PCV will not deliver a consistent tidal volume. This mode is restricted to conventional ventilation and not for NPPV.

ROLE OF EXTRINSIC PEEP IN COPD PATIENT

During controlled ventilation, work of breathing is not a problem but a major risk is dynamic hyperinflation and intrinsic PEEP which can be detrimental in this patient.

Selection of Optimal PEEP

The level of extrinsic PEEP must be enough to counterbalance the pressure, causing small airway collapse (the critical closing pressure). But should not exceed the level of intrinsic PEEP (so that it does not impair expiratory flow).

Low levels of CPAP/PEEP are recommended in patients with COPD needing mechanical ventilation either conventionally or by NIPPV. Low levels of CPAP/PEEP improve gas exchange and expiration. Start PEEP around 3 - 5 cm H_2O. Extrinsic PEEP value < 5 cm H_2O needed in most cases should be below the 'intrinsic PEEP', so that further increase in the FRC should not occur.

Extrinsic PEEP should be kept less than or equal to 60-70 % of auto–PEEP since other parameters of ventilator settings will reduce auto-PEEP slowly. This low set PEEP will not be harmful to the patient and helps to trigger breaths more easily. It also ensures that the patient is comfortable and able to trigger all ventilator breaths. The role of applied PEEP to recruit alveoli in respiratory failure from obstructive disease is less than in parenchymal disease.

Advantage of Extrinsic PEEP in COPD Patient

Addition of extrinsic PEEP less than or equal to 60-85% reduces the patient's inspiratory threshold load and keeps small airways open so reducing effective airway resistance.

- To reduce inspiratory threshold load
  The intrinsic PEEP must be overcome by the inspiratory muscles before airway pressure (pressure trigger) or flow (flow trigger) changes to produce a signal to the ventilator that a patient breath is desired. If auto-PEEP is present, then to start inspiration the patient has to generate from positive end expiratory pressure (auto-PEEP) to zero then create negative pressure to initiate inspiration, which increase work of breathing due to inspiratory threshold load imposed by intrinsic PEEP. In ventilator dependent COPD patient, low levels of PEEP induces reduction of the inspiratory threshold load without affecting EFLV.
- Keep them open during expiration
  When hyperinflation is due to small airway collapse at end-expiration, as occurs in-patients with asthma and COPD, the application of extrinsic PEEP may prevent the end-expiratory collapse of small airways and can help keep the small airways open at end-expiration.

Precaution to be Taken for Selection of PEEP

Increasing dyspnoea and other signs of increasing intrathoracic pressure (e.g. decreasing blood pressure, worsening of clinical signs of hyperinflation) suggest that excessive PEEP is being provided.

VENTILATORY SETTINGS

Controlled Oxygen Therapy

COPD patients are at risk of hypoxia since they mostly have PaO_2 $< 50\text{-}55$ mmHg. First measure in this patient is to administer oxygen to avoid death from hypoxia and myocardial dysfunction. The objective is to increase the low level PaO_2 to a safe level, around 60 mmHg without arterial pH falling below 7.25. The severity of the acidosis is a better prognostic guide in these patients than absolute $PaCO_2$ value. Uncontrolled oxygen therapy is lethal in these patients, hence administration is controlled and supervised continuously.

Start inspired oxygen of 24% and can be increased slowly upto 28-30% depending on degree of hypoxia. Three types of patterns can be observed.

- Relief of hypoxaemia, with a fall in the $PaCO_2$ and with clinical improvement is seen.
- Relief of hypoxaemia, but initial increase in the $PaCO_2$ producing falls of pH. This rise in $PaCO_2$ is seen after starting of oxygen therapy and mostly associated with drowsiness, unresponsiveness and confusion. After sometime there is a gradual fall of $PaCO_2$ to pretreatment level or lower levels, and returning pH towards normal, which is occurring with clinical improvement.
- Relief of hypoxaemia, but rapid, progressive rise in the $PaCO_2$ with fall of pH < 7.25 (with an increasing respiratory acidosis). This response is seen if oxygen is administered in an uncontrolled manner in high concentration or even with controlled and supervised oxygen therapy between 24-28 %. In these type of patients high $PaCO_2$ (65-79 mmHg) is present before starting treatment and further rise in $PaCO_2$ and lowering in arterial pH can prove lethal and dangerous. In these patients prime importance is to relieve hypoxia.

Small Tidal Volume

Small tidal volumes of 300-400 ml are used with minute ventilation not exceeding 5-6 lit/min. The FiO_2 is increased to 50-60% to correct hypoxia due to a small tidal volume. A small tidal volume prevents overinflation, preventing a further increase in intrinsic PEEP. Low tidal volume also leads to lower peak inflation pressures, reducing the risk of barotrauma, which is of great benefit to the patient.

Since high peak inflation pressures are transmitted to the intrapleural space, causing sharp reduction in the venous return, and decrease in cardiac output and can produce severe hypotension. A tidal volume should reduce more if there is a fall in arterial pressure. The blood pressure can be raised by volume expansion or by using ionotropic support. Once hypoxia is corrected by high FiO_2, an increase in $PaCO_2$ may not be dangerous.

Trigger Sensitivity

The optimal triggering threshold is difficult in COPD patient's due to intrinsic PEEP. This is because the patient has to generate a negative pressure, at least equal to the level of intrinsic PEEP before reaching trigger sensitivity level. In the presence of high intrinsic PEEP, increased inspiratory efforts are needed, which can lead to patient ventilator dyssynchrony. If the trigger sensitivity is set very low, the ventilator could be triggered very frequently and inappropriately.

Inspiratory Flow Rate and I:E Ratio

The flow rates used in such patients are usually between 40-60 lit/min, and the I:E ratio is initially set at 1:2 or 1:3. This I:E ratio allows good distribution of inspired gas allowing time for expiration.

Precautions While Setting Ventilator Parameters

Ventilator adjustment should be done carefully to bring $PaCO_2$ slowly over 24-48 hours. It is unnecessary to reduce $PaCO_2 < 50$ mmHg unless baseline is normal. A sudden decrease in $PaCO_2$ will shift respiratory acidosis to metabolic alkalosis which precipitate sudden arrhythmias and can cause sudden death.

Timing for Tracheostomy

An elective tracheostomy is preferred if ventilator support needs to be extended for more than 7 days or the patient has copious secretion that cannot be suctioned through the endotracheal tube.

GOALS OF PHARMACOLOGICAL STRATEGY IN COPD

Treat Infection of Lungs

The commonest cause of exacerbation in COPD is respiratory infection. Antibiotics play a vital role, since control of infections leads to improvement in lung function, and reverse acute respiratory failure. The antibiotic treatment is same as in status asthmaticus. Amoxicillin or amoxicillin + clavulanic acid to cover gram-positive infections and gentamicin or ciprofloxacin for gram-negative ones are preferred antibiotics. If the clinical features persist, change the antibiotics. Alternative choice of antibiotics is also necessary depending on bacterial cultures from bronchial secretions, sputum and blood. The commonest organisms which are implicated in exacerbation are *Streptococcus pneumoniae, H. influenza, Morexalla* etc.

Adequate Vascular Filling Pressures are Needed

Adequate hydration is needed. After the patient is put on the ventilator a drop in venous return is common which usually responds to volume replacement. Air trapping and resulting high intrathoracic pressures may compromise right ventricular function. Care must be taken to ensure adequate vascular filling pressures to optimise right ventricular function.

Treat Bronchospasm

Use bronchodilators, glucocorticoids and mast cells stabilisers to bring bronchospasm under the control and also promote mucociliary clearance and bronchopulmonary hygiene.

- *Bronchodilators*

  Guidelines recommend that inhaled bronchodilators should be used as first line therapy in the management of COPD and discourage the use of systemic corticosteroids. In COPD either metered dose inhaler or nebulization is beneficial. To start with, the optimal dose of salbutamol 5 mg in 4 ml normal saline, nebulized 4 hourly or more frequently depending on severity, and as improvement occurs frequency can be reduced.

  Short acting bronchodilators, beta agonists and anticholinergics (ipratropium bromide) or combination therapies can be used. Ipratropium bromide 0.5 mg 12 hourly is also nebulized routinely in these patients. If a patient has been on long term use of theophyllines, it is best not to use loading dose and intravenous aminophylline (maintenance dose - 0.5-0.6 mg/kg/hr) in a slow drip given over 24 hours. Serum levels of theophyllines help to adjust the dosage.

- *Corticosteroids*

  Oral or intravenous corticosteroids can be used in patients with acute or chronic respiratory failure due to airway obstruction. Prednisolone 40-60 mg orally, or intravenous hydrocortisone 200 mg 8 hourly, is used till improvement occurs. The dose is then gradually tapered off over 8-15 days and inhaled corticosteroids, budecort 1 mg nebulized 12 hourly is substituted.

Physiotherapy

Physiotherapy is strongly recommended in COPD patients, since patients are having excess, copious secretions. Physiotherapy consists of liquefying viscid secretions with steam inhalation, the use of nebulization, deep breathing and promotion of effective coughing. Fall in oxygen saturation is seen after chest percussion in COPD patient and it should not be done. The patient may have to wake up at night to help them, to take deep breaths, cough and expectorate since they tend to hypoventilates and retain secretions during sleep.

Weaning strategies

Weaning is like balancing on a tight rope. Continuation of unnecessary ventilation carries risk of ventilator dependency and other complications. Reintubation should be avoided because it carries 6 to 12 fold increased risk of mortality. Weaning failure is associated with increased work of breathing. Dynamic hyperinflation is a major determinant of weaning failure. Before deciding to wean the patient, infection has to be controlled, airway obstruction has to be decreased and ventilation and perfusion abnormalities to be rectified:

The problem leading to acute respiratory failure and if new problems have arisen during ventilatory support, must be under control before starting the weaning process. It is important to remember that decrease in ventilatory support, especially in older patients or patients with cardiovascular problems, might lead to early cardiovascular failure. This is seen more if there is fluid overload. Start therapy with diuretics and check serum potassium. Intravascular volume depletion should not be too fast, it can be detrimental since it may reduce preload.

Weaning should be done cautiously. For successful weaning three things are to be remembered:

- Take it easy
- Do not push
- Things take time

Decreasing the breathing load and increasing respiratory muscle capacity are the major strategies for achieving successful weaning. The measurement of P0.1 and breathing pattern may help to discover why some patients are ventilator dependent. NPPV weaning was associated with a shorter duration of invasive ventilation. It is important to note that protocols and weaning parameters are meant to guide and expedite and they never should substitute for common sense.

FAILURE AND SUCCESS OF MECHANICAL VENTILATION

Though complications are similar to other patients, dynamic hyperinflation is a major factor for ventilator dependency and weaning failure. In the presence of dynamic hyperinflation, high alveolar pressures ensue and may cause pneumothorax, ventilator induced lung injury or hypotension.

COPD patients should not be weaned from SIMV alone. The increase in resistive work due to the tracheal tube makes it difficult to unload the work of breathing. Nutritional assessment is considered as a necessary monitoring aspect during the weaning process.

MONITORING

The $PaCO_2$ level should be decreased only to the level maintained at baseline. In many patients it is around 50-60 mmHg baseline or that required maintaining a near normal $pH > 7.3$. Adequacy of oxygenation can be judged from SpO_2, respiratory rate and heart rate. Respiratory mechanics are commonly monitored to recognise any asynchrony, inadequacy in flow, excessive pressures, or inadequate volumes. It is common to experience difficulty in weaning in COPD patients from mechanical ventilation.

CHALLENGES IN VENTILATING COPD

Treatment using a ventilator is not a treatment of COPD but only a means, to buy time for other treatments to be effective. If the condition can be treated then the fear that the patient will become ventilator dependent is not true. In patients with airway disease, respiratory failure is often the end result of progressive worsening of airflow obstruction over several days. With irreversible underlying pulmonary disorders, such as COPD patients, when appropriately managed on mechanical ventilation will return to their baseline dyspnoea, increased work of breathing and increased ventilatory drive. Blood gas parameters are less useful in these cases.

Increased morbidity in mechanically ventilated COPD patients are associated with:

- *Barotrauma*
- *Fluid, electrolyte and acid base disturbances*

  Respiratory acidosis with hypokalaemic metabolic alkalosis being the commonest complications. Due to salt and water retention, there will retention of fluid. Potassium deficiency deficit may be due to β_2 agonist, theophyllines, corticosteroids, and diuretics. This loss can be slowly corrected by a slow intravenous infusion of 20-60 mEq of potassium chloride in dextrose.

- *Pulmonary infections which complicate exacerbation and crisis*
- *Cardiac arrhythmias*

  Supraventricular tachycardia and atrial fibrillation are common. The mortality is significantly higher in patients having arrhythmias. Ventilator adjustment should be done carefully to bring $PaCO_2$ slowly over 24-48 hours. It is unnecessary to reduce $PaCO_2$ <50 mmHg unless baseline was normal. A sudden decrease in $PaCO_2$ will shift respiratory acidosis to metabolic alkalosis which can precipitate arrhythmias and cause sudden death.
- *Haemodynamic instability and right sided heart failure*

  Hypotension is common if there is marked intrinsic PEEP. Patients with COPD mostly have pulmonary hypertension. An acute exacerbation of COPD can worsen pulmonary hypertension due to a variety of causes:
  - Presence of intrinsic PEEP
  - Dynamic hyperinflation
  - Hypoxic pulmonary vasoconstriction response.

  Increase in pulmonary artery pressure leads to severe right ventricular dysfunction, right sided failure, with a fall in both systolic and mean arterial blood pressure. An intravenous fluid expands intravascular pulmonary bed and improves right ventricular pump function. If fluid challenge fails to improve, vasopressors have to be started. Dobutamine is to be preferred inotropes in these patients. In older patients associated with poor left ventricular function, inotropes are necessary. Hypoxia is to be urgently corrected since it will worsen myocardial function.
- *Pneumothorax*

  The complication patients may get due to insertion of central lines or due to barotraumas. Poor response to therapy or sudden deterioration should suspect pneumothorax.
- *Nosocomial infection*
- *Aspiration*
- *Difficulty in weaning*
- *Pulmonary thromboembolism*

  This is treated with intravneous heparin
- *Gastrointestinal bleeds*
- *Mental depression*

  This is mostly seen if there is frequent admission in the ICU.

CHAPTER

40

Ventilatory Strategy for ARDS

ARDS is characterised by diffuse pulmonary microvascular injury resulting in increased permeability, thereby producing non-cardiogenic pulmonary oedema. The clinical definition of ARDS is:

- An acute onset and a history of precipitating condition
- Respiratory distress and refractory hypoxaemia not responding satisfactorily to supplement oxygen and invariably necessitating the use of mechanical ventilatory support
- Radiographic evidence of newly evolving diffuse, bilateral pulmonary infiltrates
- Pulmonary artery occlusion pressure (PAOP) < 18 mmHg (the absence of a raised pulmonary artery occlusion pressure) in the presence of normal colloid oncotic pressure.

NORMAL FLUID DYNAMICS WITHIN LUNGS – STARLING EQUATION

Fluid dynamics within the lungs are governed by the Starling equation. According to the Starling equation, the net flow of fluid across the capillaries can be summarised by the following equation:

$$Q = [\ K\ (\ P_c - P_i) - \partial\ (\Pi_c - \Pi_i\ )]$$

where,

Q – Net flow of fluid across the pulmonary capillaries

K – Permeability coefficient (it represents the endothelial leakiness for the transuding fluid)

P_c – Pulmonary capillary hydrostatic pressure

P_i – Interstitial fluid hydrostatic pressure

∂ – Reflectance (i.e. property of fluid molecules)

Π_c – Pulmonary capillary oncotic pressure

Π_i – Interstitial fluid oncotic pressure

From the above equation, it is clear that the movement of fluid between the interstitium and the pulmonary capillaries is determined by the two opposing forces, i.e. the hydrostatic pressure and the fluid oncotic pressure. Fluid oncotic pressure is due to the presence of proteins in the fluid, mainly albumin.

The summation of forces that favours transudation of fluid from the capillaries into the interstitium exceeds the forces that oppose this effect. There is an outflow of small quantities of fluid from the pulmonary capillaries into the interstium, which is mainly removed by the interstitial lymphatic.

In normal alveoli, alveolar flooding does not occur for three reasons:

- Interstium fluid is promptly removed by lymphatic
- Alveolar epithelium is leak proof by intercellular tight junctions preventing leaking of fluid into the alveoli
- Oncotic pressure within capillaries hampers outflow of fluid

PATHOPHYSIOLOGY (FIG. 40.1)

The basic pathology of ARDS is a diffuse inflammatory process that involves both lungs, and due to the loss of the integrity of alveoli – capillary membrane, protein rich fluid leaks from the capillaries into the interstium. The presence of fluid in the interstium diminishes the oncotic pressure (protective mechanism) and the interstium gets flooded with water. Thus, the lymphatics are unable to cope with this excess of fluid. The damage to the alveolar capillary membrane, results in an early interstitial and alveolar oedema leading to alveolar atelectasis.

Exudative Phase

Activation of Circulating Neutrophils

The lung consolidation in ARDS is believed to originate from a systemic activation of circulating neutrophils. The activated neutrophils become sticky and adhere to the vascular endothelium in the pulmonary capillaries.

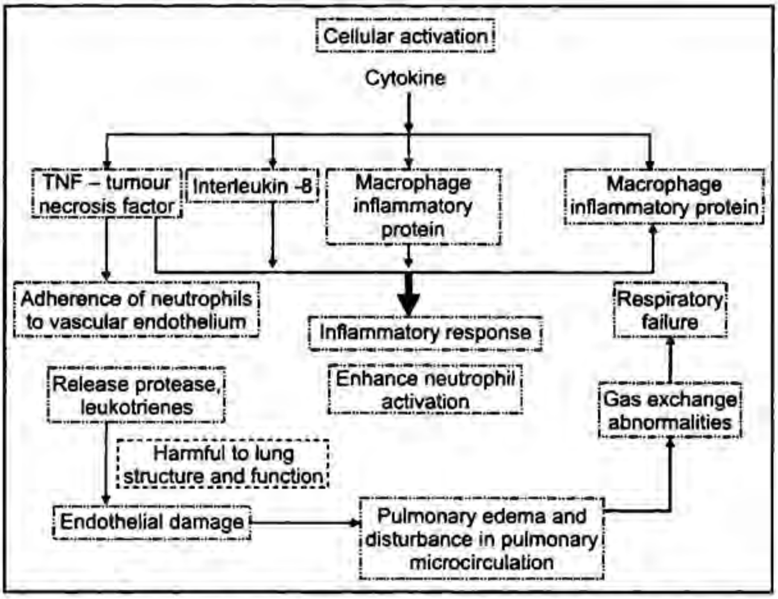
Cellular activation
Cytokine
TNF – tumour necrosis factor
Interleukin -8
Macrophage inflammatory protein
Macrophage inflammatory protein
Adherence of neutrophils to vascular endothelium
Inflammatory response
Respiratory failure
Release protease, leukotrienes
Enhance neutrophil activation
Gas exchange abnormalities
Harmful to lung structure and function
Endothelial damage
Pulmonary edema and disturbance in pulmonary microcirculation

Figure 40.1: Role of cytokines, neutrophils and coagulation system

Leaky Capillaries

Proteolytic enzymes and toxic oxygen metabolites are released from cytoplasmic granules, thereby damaging the endothelium giving rise to leaky capillaries. Neutrophils and other inflammatory mediators can gain access to the lung parenchyma and carry on the inflammatory process through leaky capillaries.

Proliferative Phase

There is a proliferation of inflammatory cells, i.e. lymphocytes and type II pneumocytes. Such inflammation then causes lung injury. Type II alveolar cells (surfactant producing cells) are destroyed leading to diminished production of surfactant, whereas the exuded proteins and inflammatory products compromise the viability of the remaining surfactant. Since the surfactant plays an important roles, the loss of functional surfactant increases the surface tension, thereby contributing to alveolar flooding, increased tissue elastance, small airway closure and atelectasis, particularly at low lung volumes.

The lung parenchymal airspaces are partially occupied by proteinaceous oedema and cellular infiltrate giving rise to non-cardiogenic (high permeability) oedema. As the condition progresses, this exudative material accumulates and eventually obliterates the alveolar airspaces. In other words, the patient will have varying degrees of interstitial oedema, alveolar flooding, surfactant dysfunction and small alveolar dysfunction. Almost 50% of the lung is airless at end of expiration. However, the exact percentage of airless and aerated tissue depends on the etiology, disease stage and severity.

Filling up of airspaces, thus severely compromises the gas exchange. Areas of lung tissue become atelectatic and poorly ventilated. The atelectatic lung unit resists the expansion and the overall lung compliance falls. The closure of alveoli means fewer alveoli are now available for gas exchange, in other words smaller lung (*baby lung*) for ventilation. Small tidal volumes are required for ventilation of these so called baby lungs.

There is an occurrence of hypoxia, direct vascular compression by raised intrathoracic pressure due to mechanical ventilation and pulmonary hypertension. It is uncertain that hypoxic vasoconstriction actually improves V/Q matching by the closing down of the blood supply to atelectatic air units (compensatory response).

Fibroproliferative Phase

This phase is followed by a proliferative phase characterised by fibroblasts laying down fibrous tissue that strangles alveoli and further reduces pulmonary compliances. Transition to this phase is not clear and can happen in association with acute lung injury.

In this phase, there is chronic fibroproliferative lung disease of variable extent. Fibrosis may start as early as the 7th day of ARDS. Patients in this phase may die of progressive intractable respiratory failure or non-pulmonary causes, because of multiorgan involvement.

CLINICAL COURSE

The clinical course of ARDS tends to follow a typical pattern, with the following four phases:

Acute Injury

Clinical findings in the early phase are related to the primary insult. There may not be an apparent pulmonary dysfunction unless the lungs are primarily involved. The chest x-ray can be unrevealing in the first few hours of the illness.

Latent Period

For approximately 6 to 72 hours after acute injury, the patient appears to be stable or recovering. Some patients hyperventilate with a respiratory alkalosis despite adequate PaO_2 values, and the chest X-ray film may show fine bilateral reticular infiltrates, consistent with the increased interstitial fluid. The infiltrates may be more prominent in the peripheral lung fields, like the pattern of infiltration.

Acute Respiratory Failure

In the early stage, a slight but disproportionate tachypnoea may be the only warning sign. The chest X-ray may be normal with crepitations. The early and important sign during the early stage is a fall in the PaO_2 and increased alveolar arterial oxygen gradient. Clinical diagnosis of acute lung injury should be considered if the patient who has had an initiating insult, subsequently develops tachypnoea, dyspnoea, and hypoxaemia refractory resulting in increased inspired oxygen concentration, diffuse infiltrates on chest X-ray and decreased pulmonary compliance. These changes may have a rapid onset. Congestive heart failure must be ruled out as an acute cause of pulmonary oedema.

Diffuse crepitations may be noted on auscultation and the chest X-ray demonstrates diffuse, hazy bilateral infiltrates due to interstitial and intra-alveolar fluid with no cardiomegaly. Decreased pulmonary compliance manifests as tachypnoea and dyspnoea requiring ventilatory support as cyanosis and desaturation appears on pulse oximetry despite administration of maximal inspired oxygen concentration. During this phase, some patients may succumb to death, some may recover and others may have progressive pulmonary dysfunction with transition to the next phase.

Multi-organ Failure (Fig. 40.2)

As the respiratory failure worsens, one or more other organs may show sign of dysfunction and failure. The changes in the pulmonary endothelium mirroring widespread endothelial changes in other organs also. This phase is characterised by the ventilation perfusion abnormalities, impaired coagulation profile and unstable cardiovascular system as a part of worsening systemic inflammatory response syndrome (SIRS), which may progress to multi-organ dysfunction syndrome/ failure.

Multi-organ dysfunction is due to direct damage by inflammatory mediators, as well as, tissue ischaemia due to insufficient oxygen delivery in the presence of increased metabolic demand. Progression to the mechanical ventilation often occurs in the first 48 hours or the illness.

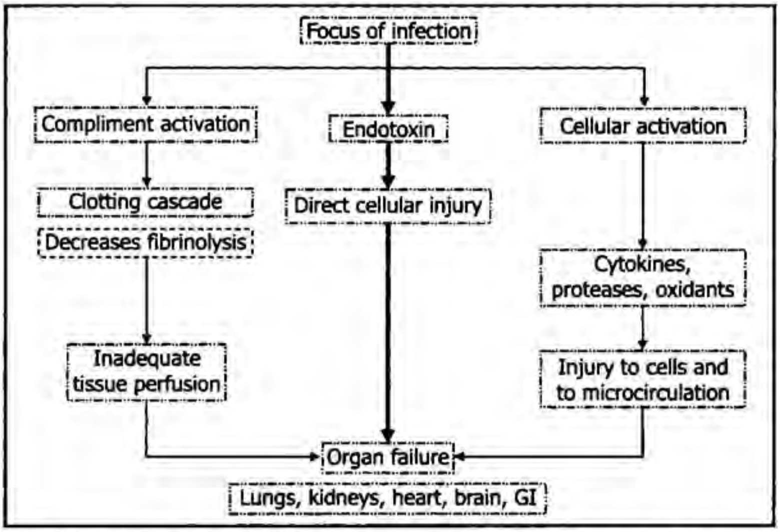

Figure 40.2: Pathogenesis of acute lung injury and organ failure

COMMON PREDISPOSING CONDITIONS/RISK FACTORS TO ARDS

The following are the risk factors to ARDS:

Direct Injury Risk Factors

- Aspiration
- Near drowning
- Toxic gas inhalation
- Pulmonary contusion/Pneumonia
- Diffuse pulmonary infection - bacterial and/or viral

Indirect Injury Risk Factors

- Long bone fracture
- Cardiopulmonary bypass
- Blood products, catheter sepsis, drugs
- Massive transfusion from emergency resuscitation
- Intracranial hypertension
- Pancreatitis
- Amniotic fluid embolism
- Poisoning

Patient's with tropical problem

- Tetanus
- Leptospirosis

- Cerebral malaria
- Rabies

It is already mentioned that the common feature in these conditions is the activation of the neutrophils in the pulmonary or systemic circulation. Thus, any condition that produces a systemic inflammatory response can predispose to ARDS. The most common predisposing condition is sepsis, which is a systemic inflammatory response (fever or leukocytosis) due to an infection.

ARDS AND ACUTE LUNG INJURY (ALI) CAN BE DIVIDED FURTHER

- *Lung specific injuries*: Massive aspiration, bilateral pneumonias, drowning
- *Lung injuries as a part of systemic processes*: Sepsis, pancreatitis (i.e. the lung is one of many organs affected by a diffuse inflammatory response called systemic inflammatory response syndrome [SIRS])
- *More focal parenchymal injuries including*: lobar pneumonias, regional aspiration and lung contusions.

ARDS and ALI syndrome are also diffuse parenchymal injuries that have specific diagnostic criteria.

Parameter	ARDS
Onset	Acute
Clinical condition	Predisposing condition
Chest X-ray	Bilateral infiltrates
Wedge pressure	<18 mmHg
Gas exchange	In ARDS-PaO_2/FiO_2 less than or equal to 200 mmHg ALI-PaO_2/FiO_2 ratio is less than 300 Normal lung-PaO_2/FiO_2 ratio is more than 300

Acute lung injury when mild to moderate is termed acute lung injury and when severe is termed the acute respiratory distress syndrome. The degree of severity of injury being assessed by the PaO_2/FiO_2.

DIAGNOSTIC DILEMMAS

The distinction between inflammation and infection is an important one because inflammation, not infection, produces ARDS. The other conditions that can mimic the clinical presentation of ARDS include pneumonia, acute pulmonary embolism, and cardiogenic (hydrostatic) pulmonary oedema.

Overlapping features of ARDS and other causes of acute respiratory failure

Feature	*ARDS*	*Left heart failure*	*Pneumonia*	*Pulmonary embolism*
Fever, leukocytosis	Yes	Possible	Yes	Yes
Bilateral infiltrates	Yes	Yes	Possible	Unlikely
Pleural effusions	Unlikely	Yes	Possible	Possible
Wedge	Normal	High	Normal	Normal
Lung lavage protein	High	Low	High	High

PHYSICAL EXAMINATION

The physical findings in ARDS (e.g. fever, tachypnoea, and rales) are non-specific, and can be seen in many conditions. Fever can be also feature of cardiogenic pulmonary oedema, i.e. oedema resulting from an acute myocardial infarction or inflammatory myocarditis.

How to Differentiate between ARDS and Cardiogenic Pulmonary Oedema

Comparison of severity of the hypoxaemia and chest X-ray

The severity of the hypoxaemia can sometimes help to distinguish ARDS from cardiogenic pulmonary oedema. In the early stages of ARDS, the hypoxaemia is often more prominent than the chest X-ray abnormalities. In the early stages of cardiogenic pulmonary oedema, the X-ray abnormalities are often more prominent than the hypoxaemia.

However, there are exceptions, and severe hypoxaemia can occur in cardiogenic pulmonary oedema, if the mixed venous oxygen pressure ($P\bar{V}O_2$) is reduced from a low cardiac output. When a chest X-ray shows bilateral pulmonary infiltrates, the principal concern is to differentiate ARDS from cardiogenic pulmonary oedema, which is a difficult task. Thus, chest X-rays are not reliable in distinguishing ARDS from cardiogenic pulmonary oedema.

Predisposing Conditions

The presence of high-risk conditions for ARDS may be the best means of distinguishing ARDS from cardiogenic oedema. However, many of the conditions that predispose to ARDS also predispose to pneumonia and pulmonary embolism. So the predictive value of these conditions is limited if pneumonia or pulmonary embolism is suspected.

Wedge Pressure

The standard method of distinguishing ARDS from cardiogenic pulmonary oedema is to measure the pulmonary capillary wedge pressure (PCWP) with a pulmonary artery catheter.

PCWP < 18 mmHg = ARDS

PCWP > 18 mmHg = Cardiogenic pulmonary oedema

Broncho-alveolar Lavage

Bronchoscopy is generally not reasonable as a diagnostic tool in ARDS. It may be performed rarely for other reasons in a patient with suspected ARDS (e.g. to identify infection). A bronchoalveolar lavage can be performed safely in patients with ARDS during bronchoscopy.

Neutrophils: In normal subjects, neutrophils make up less than 5% of the cells recovered in lung lavage fluid, whereas in patients with ARDS, as many as 80% of the recovered cells are neutrophils. The neutrophil preponderance in lung lavage fluid can help to distinguish from cardiogenic oedema.

Protein concentration: The protein concentration in oedema fluid and blood can be used to distinguish watery hydrostatic oedema from inflammatory oedema.

Protein (oedema/serum) < 0.5 = Hydrostatic oedema
Protein (oedema/serum) > 0.7 = Inflammatory oedema

Neutrophils and protein concentration may help to distinguish ARDS from cardiogenic oedema, but may not be helpful in patients with pneumonia.

Pathophysiological Features Relevant to Ventilatory Support

Lungs of patients with ARDS are mechanically heterogeneous and vary enormously in their patterns of infiltration, their inflation, and collapse properties and their regional pathology. ARDS is an inflammatory process and not an accumulation of watery oedema fluid (not type of pulmonary oedema), giving rise to both mechanical and gas exchange abnormalities. This is an important distinction for the diagnosis and management of ARDS.

Mechanical Properties of the Injured Lung

The physiological changes include a reduced functional residual capacity, reduced pulmonary compliance (stiff lungs) increased ventilation perfusion inequalities, an increase in the right to left shunt within the lungs and an increase in the extravascular water content of lung but with normal capillary wedge pressure. All these changes are due to the pathological abnormalities to the damage of alveolar capillary membrane with increased capillary permeability, leading to oedema, inflammation and subsequent fibrosis.

Decreased Lung Compliance

Replacement of airspaces by inflammatory debris, cells and fluid, results in a lung whose aerated capacity and compliance is severely reduced. The compliance of the respiratory system (lung and chest wall) falls during ARDS for two reasons:

- Infiltrated and surfactant depleted tissue is inherently more elastic than healthy alveoli
- Many functional lung units operate near their elastic limits because fewer units are available to accept the tidal volume.

Increased Work of Breathing

Under normal conditions, surfactant modified surface forces allow the lung to inflate and deflate at similar pressures. In an injured lung there is reduced aeratable capacity due to surfactant depletion. Hence for the same tidal volume, the mechanical breathing work increases dramatically.

Amplification of the Stresses of High Pressure at the Junctions of Closed and Open Lung Units

For any specified airway pressure, there exist lung units that are closed and those that are open.

- *Local in-flammatory response:*
  Due to nonphysiologic stretching forces. In open units, lung units exist in different states of expansion depending on the local transthoracic pressures that distend them. Even at airway pressures, that are generally considered modest, some of the open lung units are verge on overdistension, whereas, the others are on the compliant portion of their pressure volume relationship.
  Alveolar distension that approaches the elastic limit stimulates surfactant production. The repetitive application of nonphysiologic stretching forces during high tidal volume/high pressure ventilation is currently believed to initiate molecular signaling of a local inflammatory response.
- *Amplification of shear forces (Fig. 40.3):*
  Heterogeneously affected lung amplifies the stresses of high pressure at the junctions of closed and open lung units in a nonlinear fashion.

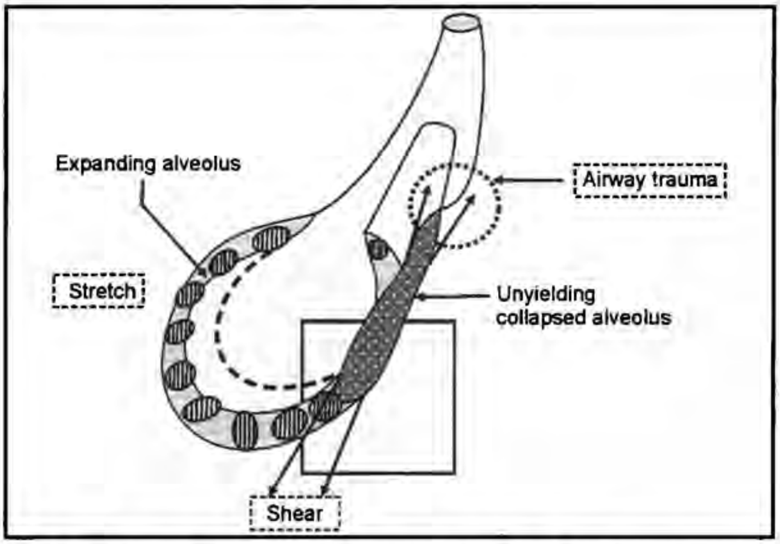

Figure 40.3: Stress amplification at the junction of open and closed lung units

Such recurring forces not only strain the lung's structural meshwork to signal inflammation, but also assist in reopening potentially recruitable (atelectatic) lung units. When atelectatic tissue are subjected to high pressures, shearing forces within these zones are amplified and damage may be of sufficient magnitude to tear the delicate terminal airways or alveoli themselves, creating microwounds that produce tissue haemorrhage and provoke inflammation as a secondary phenomenon.

Stress amplification at the margins of dissimilar tissues is at least partially a function of their relative volumes. (A flooded, gasless alveolus of volume similar to its air-filled alveoli may experience forces that are no more intense). Whether injury results from the process of repetitive popping open of lung units under high pressure or simply from the cumulative result of high tension stretching at the interface remains to be resolved. Thus it appears therefore that there are at least two critical elements that place the injured lung at risk for ventilation associated lung damage, i.e. high inflating pressure and the prevalence of collapsed lung units that interfere with open ones.

- *Reopening of Lung Units by Redistributing Alveolar Liquid*
  Reopening of lung units by alveolar pressure may occur primarily by redistributing alveolar liquid and shifting fluid volume from the alveolar to the interstitial compartments of the lung not by atelectasis reversal (recruitment). At the microscopic level, lung units collapse can be divided in two broad categories, i.e loose and sticky atelectasis.

- **Loose atelectasis**

  Loose atelectasis arises primarily from compressive forces of the heavy lung acting to close small airways and responds to relatively low levels of transpulmonary pressure.

- **Adhesive or sticky atelectasis**

  Adhesive or sticky atelectasis results from gas absorption and requires very high pressure to reverse. High concentration of inspired oxygen encourages gas absorption in poorly ventilated regions, as well as interferes with surfactant kinetics. These hard-to-recruit units may be among the first to close as high airway pressure is withdrawn.

  In the settings of severe ARDS, pressures that exceed 60 cm H_2O may be needed to complete the recruiting process. Although opening of lung units is primarily a function of transalveolar pressure, the duration with which pressure applied also contributes, that is, a lower pressure may be sufficient if applied for an adequate period. Moreover, atelectatic lung units tend to 'yield' (open) in a irregular fashion, with high sustained pressure causing a serial snapping open of small blocks of units. Irregular lung opening during the tidal cycle may give rise to audible crackles, usually best heard in dependent regions.

- *Degree of Ventilation Perfusion (V/Q) Mismatch and Shunt may Depend on the Phase of Ventilation*

  Also, the degree of V/Q mismatch may change during the ventilatory cycle. If cyclical alveolar collapse occurs, there may be low V/Q relationship with inspiration and shunting during expiration. Increase in dead space (V/Q = ~) is not a usual manifestation of parenchymal lung disease, unless there is very severe end-stage injury. That is why in parenchymal disease, hypoxaemia tends to be more of a problem than CO_2 clearance.

- *Effect of Heterogenecity in Distribution of Gas*

  However, in all but the most diffuse diseases (e.g. cardiogenic oedema), there can be marked regional differences in the degree of inflammation present and corresponding mechanical abnormalities. This heterogeneity can have significant impact on the effects of particular mechanical ventilation strategy leading to regional overdistension injury for normal lung.

  This is because delivered gases preferentially go to the regions with the highest compliance and the lowest resistance (i.e. the more normal regions) rather than to more affected regions. A "normal-sized" tidal volume thus may be distributed preferentially to the healthier regions and may result in a much higher regional tidal volume with potential regional overdistension injury. One of the rationales for using pressure-limited ventilation in severe parenchymal injury is that, it limits the maximal distension in all units to the set level, regardless of changes in regional lung mechanisms.

- *Alveoli are Prone for Oedema Formation*
  The increased microvascular permeability of ALI/ARDS renders it vulnerable to oedema formation and impedes its relative rate of oedema clearance.

Pulmonary Hypertension

Pulmonary hypertension causes an increase in microvascular filtration pressure with an increase in alveolar interstitial lung oedema and may also lead to right heart failure. In parenchymal injury, severe hypoxaemia coupled with direct vascular injury often leads to high pulmonary artery pressures. This can compound the gas exchange problem overloading the right ventricle and reducing perfusion through the lung. The need for adequate right heart filling pressures must be balanced against the risk of worsening lung oedema when fluid therapy and ventilator pressures are adjusted.

Cyst Formation

Parenchymal injury also can affect the airways, especially all the bronchioles and alveolar ducts. These narrowed and collapsible small airways can contribute to reduced regional ventilation to injured lung units. This also can lead to regions of air trapping and it may lead to subsequent cyst formation during the healing phase.

INVESTIGATIONS

Chest X-ray

In the early stage, chest radiograph shows interstitial oedema and in later stages a full blown pulmonary oedema is seen. The pulmonary oedema usually causes bilaterally symmetrical shadowing, but the shadowing may be initially predominantly unilateral, depending on the position of the patient. The absence of cardiomegaly, Kerley's lines and vascular redistribution towards the upper lobe, helps to distinguish ARDS pattern from cardiogenic pulmonary oedema.

Arterial Blood Gases

In the initial stages, varying degrees of hypoxia is usually seen with hypocapnia. However in the late stage, hypercapnia may be seen.

The pH will show respiratory alkalosis in early stages and respiratory acidosis and metabolic alkalosis in the later stage. Refractory hypoxia indicates increased right to left shunt and dead space ventilation in addition to ventilation perfusion of mismatch.

Haemodynamic Measurements

Pulmonary oedema with pulmonary wedge pressure <18 mmHg in the presence of a normal colloid oncotic pressure is diagnostic of acute lung injury.

Pulmonary Compliance

Lung compliance is usually decreased to < 30 ml/cmH_2O.

Routine Tests

Renal, hepatic and haematological parameters to be done since ARDS are multi system disease.

Full Bacteriological Screening

Culture of blood, tracheal aspirates, and urine is necessary.

PROGNOSIS

The prognosis of acute lung injury depends on the following factors:
- Nature and severity of the precipitating factor
- Severity of lung injury
- Presence and degree of dysfunction of other system
- Associated diseases.

INDICATIONS OF VENTILATORY SUPPORT

- Refractory hypoxaemia
- Excessive work of breathing
- Respiratory rate > 30/min
- Minute ventilation > 12 lit/min
- Haemodynamic instability
- Inability to protect airway
- Anticipated rapid clinical deterioration.

GENERAL OBJECTIVES FOR VENTILATORY SUPPORT

- Treat the cause
- Ensure adequate oxygenation of arterial blood
- Provide sufficient oxygen transport to vital organs and tissues

- Assist in eliminating carbon dioxide
- Relieve excessive burden placed on the respiratory muscles
- Help to maintain alveolar stability
- Facilitate therapeutic measures where ventilatory control is required.

The assignment of ventilation safety in patients with injured lung is made more difficult by the mechanical heterogeneity of the respiratory system and diversity of pathophysiology.

- The chest wall also influences regional lung volumes and recruitability.
- Risk for ventilatory induced lung injury (VILI) is proportional to transalveolar pressure.
- Lung recruitment is essential to avoid ventilatory induced lung injury (VILI).

GENERAL GUIDELINES FOR VENTILAT[illegible]

1. *The patient's comfort and safety* is the first priority. Adjust ventilator parameter empirically depending on clinical norms rather than fixed formulation of rules.
2. *Noninvasive mechanical ventilation* using a comfortable face mask may overcome short-lived deficits of oxygen exchange without the need for intubation. However in practice, there is a need to control airway, apply high level of end-expiratory pressure and to sustain support for extended periods usually prohibit the use of noninvasive ventilation.
3. *Prevention of mechanical trauma* is the most important priority than maintenance of normocapnia or the avoidance of oxygen toxicity.
   - Upper acceptable limits of plateau pressure cannot be specified
   - Very high FiO_2 can be a risk for oxygen toxicity and absorption atelectasis. FiO_2 should be less than 0.7.
4. *Consider the impact of chest wall stiffness* (including abdominal contents) on transpulmonary pressure and gas exchange efficiency (Fig. 40.4). Decreased the chest wall compliance and increases the transpulmonary pressure required to inflate the lung. Any specified pressure is associated with less transpulmonary pressure in the presence of stiff chest wall or expiratory effort.
5. *In severe cases, try to minimizse ventilatory demands,* which indirectly help to reduce airway pressures, high rates of gas flow and cardiac output requirements.
6. *Monitor haemodynamics* as well as respiratory mechanics and gas exchange efficiency when regulating ventilatory therapy.

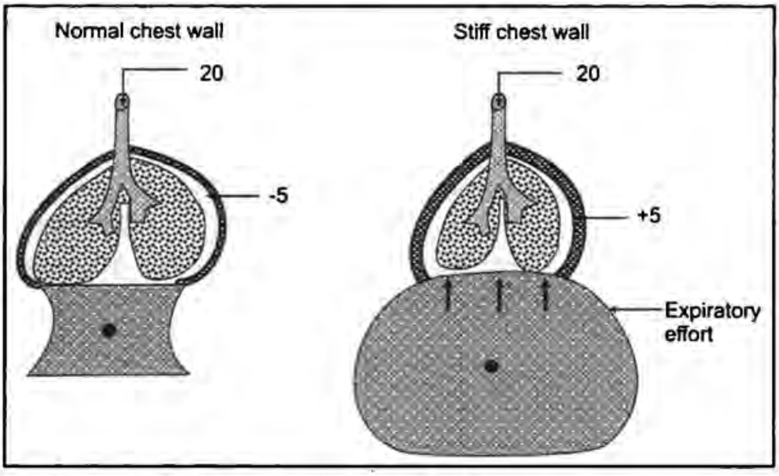

Figure 40.4: Effects of stiff chest wall on transpulmonary pressure

7. *PVC, PSV, BiPAP, APRV-pressure limited ventilation are preferred* for varieties of benefit. Volume control mode of ventilation can be used to assess respiratory mechanics and lung's gas exchanging efficiency for CO_2.
8. *Incorporate the 'challenge principle'* in making therapeutic decisions regarding both the intensification and the withdrawal of therapeutic measures.
   - Recruiting maneuver to assess lung unit instability.
   - Closely monitored challenges of fluid administration or removal
9. *Prone position is indicated* (unless contraindicated) if high ventilatory pressures, PEEP, and FiO_2 are needed to maintain adequate supine arterial O_2 tension. Mildest cases and patients which are improving should be excluded.

POINTS TO BE REMEMBERED—IN INITIAL PHASE OF STABILIZATION AND SUPPORT

- *Always determine severity of oxygen impairment* and if it is associated with any reversible condition.
- *Always decide adequacy of noninvasive ventilation* or whether it is mandatory to intubate, which mainly depends on the condition of the patient.
- *Always come to a decision for the type of ventilation.* Control ventilation or spontaneous breathing depends on condition of the patient. Controlled or nearly controlled ventilation is recommended to suppress respiratory efforts for most severely involved patients during the early stage of support. If an inappropriate respiratory drive exists

or if patient triggering of assisted breath is uncomfortable, sedation or paralysis may be needed. Routine employment of paralysis and controlled ventilation to reduce VO_2 also should be avoided due to risk of long-term myopathy.

Generally, severe respiratory failure is managed during the acute phases with an assist/control (A/C) mode of ventilation. This ensures that all positive breaths are given by the ventilator, which helps to abolish the work of breathing of respiratory muscle. The patient can trigger breaths, which may help in controlling CO_2 and improving patient comfort.

Desired Goals – Lung Protective Strategies

- Moderate hypercapnia is well tolerated.
- The beneficial targeting priorities are towards the lung protection, maintenance of appropriate hemodynamics and oxygen delivery.
- The best strategy is to use the least PEEP and adequate tidal volume, which is needed to achieve acceptable gas exchange while avoiding alveolar collapse and reopening of unstable lung units.
- Recruiting maneuvers helps
  - To characterise PEEP responsiveness
  - To set the PEEP – tidal volume combination
  - To determine the relative staus of intravascular filling and response to altered cardiac loading conditions.

How to Start Ventilatory Support – Points to Remember

- Ventilatory effort is to be kept a minimum.
- Traditionally used sustained inflation with high pressure is no more effective and is less well tolerated haemodynamically than recruitment method.
- Recruitment method is based on pressure controlled ventilation that achieves lower mean pressure but similar peak pressure during its inspiratory phase.
- If oxygenation and lung mechanics do not improve significantly with high level of PEEP as a recruiting technique, then the patient is considered to have low recruiting potential in that position and that specific time.
  - Management goal in the recruitable group emphasises the maintenance of high level end-expiratory pressure, where as in poorly recruitable patient PEEP is maintained as low as feasible-generally in the range of 5-10 cm H_2O.
  - In both the groups, end-inspiratory plateau is kept less than 30 cm H_2O except when the chest wall compliance is very low.

Mechanical ventilation is the supportive modalities of ARDS and PEEP is a crucial part of the strategy. Physiological PEEP is the small amount of PEEP wherein 3-5 cmH_2O is applied to the ventilated patient to compensate for the loss of glottic function due to ETT tube.

Concrete Relationship between Plateau Pressure, PEEP, and Tissue Strain

In the generation of tissue strain, the driving pressure for tidal volume (the difference between plateau pressure – PEEP) acts as a force lever arm, where PEEP acts as the fulcrum.

Levels of plateau pressure and PEEP produce strain within acceptable limits (Fig. 40.5).

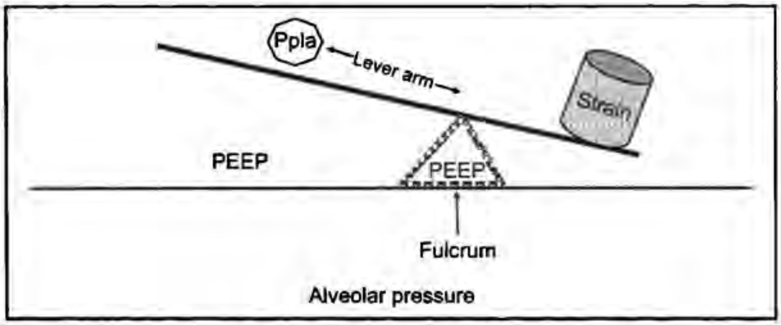

Figure 40.5: Acceptable limits of plateau pressure and PEEP

Increasing plateau pressure while keeping PEEP at a level insufficient to hold open unstable units lengthen the lever arm and produces excessive tissue strain (Fig. 40.6).

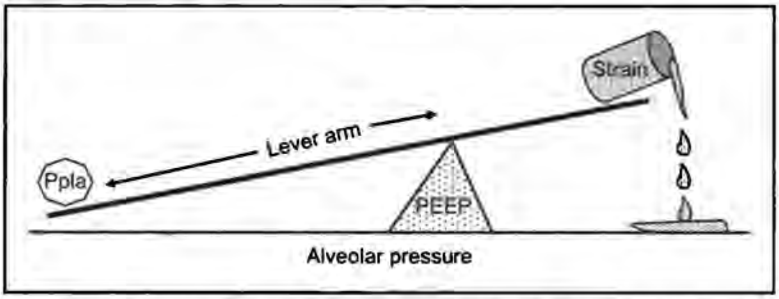

Figure 40.6: Increasing plateau pressure while keeping PEEP insufficient level

When PEEP is increased, the same high plateau pressure that caused damage previously is better tolerated, thereby fewer lung units are placed at risk after recruitment and the lever arm of driving pressure is shortened (Fig. 40.7).

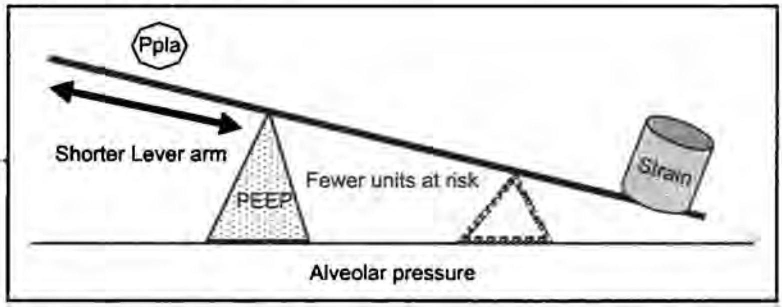

Figure 40.7: Increasing PEEP with the same high plateau pressure

How to adjust PEEP?

Patients with recruitable lung units should respond to increased PEEP and recruiting manoeuvers by demonstrating *improved alveolar mechanics* and *improved gas exchange.*

This improvement is reflected by both, i.e. increased PaO_2 and a reduction of the ratio of minute ventilation to $PaCO_2$. These beneficial changes are accompanied by only marginal effects on hemodynamics, as judged by systemic blood pressure and central venous oxygen saturation. Assuming an unchanged rate of CO_2 production, the latter index also reflects the efficiency of CO_2 elimination, which is expected to improve with recruitment and deteriorate with overdistension.

Clinical Significance of Inspiratory Crepitations in Spite of PEEP

Inspiratory crepitations audible over the dependent zones of the chest suggests that recruitment and derecruitment are occurring with each breath. This also indicates that recruitment manoeuvres and higher levels of end expiratory pressure may be needed for disappearance of the inspiratory crepitations. Crepitations occurring late in inspiration are of more concern because they may originate in units opening under relatively high pressures.

Evaluation of Response to PEEP and CO_2 Exchange

In evaluating response to PEEP, it is vital to consider both CO_2 exchange as well as oxygenation response. When PEEP is applied PaO_2 tends to increase, except in rare instances when PEEP induced impaired cardiac output causes mixed-venous O_2 content to fall. Oxygenation improvement by application of PEEP may be accounted for, either by recruitment of lung units or by reduced/redirected blood flow within the injured lung. In the latter situation, $PaCO_2$ also may rise.

Oxygenation improvement is basically due to alveolar recruitment; CO_2 exchange may also improve with this, reflecting increased alveolar ventilation. Improving of refractory hypoxaemia, in prone positioning also works under the same principle. However, there is significant improvement if small to moderate tidal volume or driving pressure is used and adequate preload and optimisation of haemodynamics is ensured. Repeteadly opening and closing during the respiratory cycle and unstable alveoli generate shear forces at their interfaces with relatively normal alveoli, thereby increasing the chances of injury in these regions (atelectrauma).

PEEP Usefulness is More in non-pulmonary Aetiologies

By increasing alveolar recruitment and increasing FRC, PEEP makes the lungs more compliant. In practice, around 15 cm H_2O PEEP has been shown to optimise the lung compliance by providing internal splinting to unstable alveoli and keeping them from collapsing during expiration. With the addition of PEEP, a proportion of closed alveoli opens up and stays open, decreasing the inhomogeneity of ventilation. PEEP seems to be more beneficial in ARDS of non-pulmonary aetiologies where lung water is confined to the interstium (in the initial stages), than in ARDS of pulmonary origin (pneumonia) where alveolar filling is major pathology.

GOALS OF TOTAL VENTILATOR SUPPORT

Goals	*Parameter*	*Threshold*
	pH	7.20-7.45
Ventilation/CO_2 clearance	The set ventilator frequency is used to control the CO_2. The willingness to accept a respiratory acidosis to prevent lung overstretch sometimes is referred as permissive hypercapnia	
Oxygenation	SaO_2	> 90 %
Avoid iatrogenic stretch injury	P_{plat}	< 30 cmH_2O with adequate recruitment
Oxidant injury	FiO_2	< 0.6

Contd...

Contd...

	Initial ventilator settings	
FiO_2	0.80	
PEEP	5-8 cmH_2O	Depending on haemodynamic tolerance
Tidal volume	6- 10 ml/kg	Depending on inspiratory plateau

Once the patient is stabilised (Generally within 10-20 minutes), the ventilatory settings are adjusted depending on the patient and guided by pulse oximetry $SaO_2 > 90\%$, patient comfort.

FiO_2

The goal and aim for oxygenation in the blood is a PaO_2 above 60 mmHg with low FiO_2 0.6 or less. At this level haemoglobin is completely saturated and PaO_2 above 60 mmHg will not serve to increase the oxygen delivery to the tissue any further. Adjustments of the inspiratory time may help to improve worsening oxygenation.

Tidal Volume is Adjusted in Such a Way

- Lungs are functionally small in ARDS; low tidal volumes are used to avoid alveolar overdistension, barotrauma and hypotension.
- To provide adequate gas exchange, maintaining sufficient tissue oxygenation, while minimising any potential iatrogenic lung injury.
- To provide the best pH and SaO_2 for the least amount of unnecessary stretch and inspired oxygen concentration (FiO_2) exposure.
- To avoid a PEEP/V_T combination that unnecessarily overdistends the lung at end inspiration, generally reflected by elevation in plateau pressure (end-inspiratory alveolar pressure) greater than approximately 30-35 cmH_2O. Generally this involves V_T of 8-10 ml/kg although V_T as low as 5 to 6 ml/kg may be needed.

Respiratory Rate

In earlier stage respiratory rate is kept high since patient is having high minute ventilation, trying to maintain reasonable $PaCO_2$ level. Increased rate decreases expiratory time and can lead to intrinsic PEEP.

Inspiratory Flow Rate (Volume Controlled Ventilation)

To increase expiratory time, inspiratory flow rate has to be increased which allows delivery of the set tidal volume within a shorter inspiratory time. However, this will increase airway pressure to some extent. Manipulations to decrease tidal volume and/or respiratory rate which will bring airway pressures to as safe levels as possible, but $PaCO_2$ should be allowed to rise in a controlled manner, since hypoventilation will be inevitable.

Decelerating waveform, the inspiratory flow gradually decreases as inspiration progresses and leads to smaller rise in airway pressure. However, slowing of waveform decreases inspiratory time and may produce air trapping by raising airway pressure.

Inspiratory Time

Inspiratory time adjustment helps patients with worsening hypoxaemia (just like. FiO_2, PEEP). Lung in ARDS is heterogenous with varying time constants and diseased alveoli will open up if inspiratory time prolongs, and will take part in ventilation. Inspiratory time can be prolonged with decreasing flow rate or decelerating wave form or inspiratory pause.

Other measures	
Estimate intravascular volume status	Arterial blood pressure, central venous pressure, urine output, urinary electrolytes
Conform adequacy of intravascular volume	Volume challenge, central venous and pulmonary artery catheter
Replace volume deficit/ support circulation	Vasopressor/inotropes
Determine the recruitment potential of the patient	Recruiting maneuver/PEEP trial
PEEP trial	Oxygenation and $PaCO_2$ change Alteration in mechanics Haemodynamic response
	Adjust PEEP and tidal volume combination to the lowest values that sustain recruitment benefit
The preferred angle for head elevation in supine patients	30° to horizontal with frequent (at least every 2-4 hours) lateral turning.

MONITORING AND STRATEGY

Ventilatory Monitoring

- Monitor plateau pressure, and indicators of compliance and resistance
- Monitor oxygenation and CO_2 excretion

Ant-MODS Monitoring and Strategy

Be especially aware of brain and kidney function and treat infectious complications. Maintain an acceptable haemoglobin level and be aware of ensuring signs of multiple organ dysfunction syndromes, by monitoring the organs at risk.

Cardiovascular Monitoring and Strategy

- *Monitor heart rate and rhythm*
- *Monitor arterial blood pressure* and try to keep it close to the patient's normal value.
- *Cardiac filling pressures*

  Central venous pressure (CVP) and wedge pressures tend to overestimate cardiac filling volumes during positive-pressure mechanical ventilation, particularly when PEEP is applied. This is partly because of transmission of intrathoracic pressures into the lumen of intrathoracic blood vessels, which increases intravascular pressure without changing transmural pressure (the pressure that determines ventricular stretch and oedema formation).

  Thus, a normal CVP or wedge pressure does not necessarily indicate normal cardiac filling volumes during positive-pressure mechanical ventilation. In this setting, CVP and wedge pressure are interpretable only if they are reduced or are less than the level of applied PEEP.

Maintain an Adequate Intravascular Volume

Intravenous fluid replacement should be given carefully and "drowning accidents" should be avoided. It is safe to combine volume substitution with early sympathomimetic support.

- *Volume infusion:*

  The volume infusion is indicated if the cardiac output is inadequate, e.g. a cardiac index below 3 $l/min/m^2$ or/and CVP or wedge pressures is not elevated.
- *Dobutamine:*

  Even though ARDS is called leaky-capillary pulmonary oedema, the lung infiltration in ARDS is inflammatory exudates, so volume infusion is not different here from volume infusion in a patient with pneumonia. If volume infusion is not indicated, dobutamine is used to augment the cardiac output.

- *Avoid dopamine:*
  Dopamine should be avoided because of its tendency to constrict pulmonary veins, which will raise the wedge pressure while reducing the left-ventricular end-diastolic volume.
- *Avoid vasodilators:*
  Vasodilators should also be avoided because of their tendency to increase intrapulmonary shunt, which can add to the primary gas exchange abnormality in ARDS (vasodilator prostaglandins are the exception).

	Prone position
Adjust tidal volume	Same values are used in the supine position. An increase in plateau pressure suggests that chest wall compliance has been altered by proning
Prone is advisable (12-20 hours day) (provided no contraindication)	Moderate to severe disease regardless of their 'recruiting test' unless patient is improving rapidly
	> 10 cmH_2O of PEEP at an FiO_2 of 0.6 to maintain O_2 saturation at 90%
	Reversion to the supine position at least once a day
Proning discontinued	No longer makes an impressive difference to oxygenation and plateau pressure can be kept in a safe range
Prone position will help	Lymphatic drainage Secretion removal Release lower lobes of the lungs from the need to support the weight of the heart
	Reverse Trendelenberg at 15-30° is preferred to flat 0° horizontal
	Prone position evenly distributes transpulmonary pressure. This even distribution of pressure not only reduces local tissue strains but effectively recruits well perfused dorsal parenchyma thereby improving oxygenation in most patients

Avoid Blood Transfusions

Transfusion is often recommended to keep the Hb above 10 g/dl, but there is no basis for this recommendation. There is a tendency for blood transfusions to cause ARDS. It is advisable to avoid transfusing blood products in ARDS. If there is no evidence of inadequate tissue oxygenation, there is no need to correct the anaemia.

Types of mode which can be used in ARDS

- Volume control — Flow controlled, time or volume cycled ventilation
- Pressure control — Pressure targeted, time cycled ventilation
- Pressure support — Pressure targeted, flow cycled ventilation
- Dual mode

Pressure Versus Volume-targeted Breaths

The choice of pressure versus volume-targeted breaths depends on which feature is required for the clinical goal.

- *CO_2 clearance is of primary concern*
  Volume targeted ventilation.
  If CO_2 clearance is of primary concern and patient and lung stretch are the less important issues, e.g. mild lung injury with a cerebral mass lesion, then volume targeted ventilation is preferable.
- *Overdistension risk is high and/or patient synchrony is needed*
  If overdistension risk is high and/or patient synchrony is more of an issue than CO_2 clearance, e.g. severe ARDS with normal cardiac and neurologic function. Pressure targeted ventilation is preferable.

Volume Control

Flow control ensures that the desired tidal volume will be delivered reliably. However, alveolar pressure can increase to a dangerous level, which is a function of delivered volume and compliance during passive ventilation. In volume control, delivered flow will not change according to patient's demand.

Pressure Control

Pressure control offers the flexibility to satisfy flow demand and under passive conditions ensures that alveolar pressure does not rise higher than set peak airway pressure. Inherent decelerating flow may improve the distribution of ventilation moderately among heterogeneous lung units compared with constant flow. The potential adverse effects of using pressure control are that delivered volume is a function of the respiratory mechanics and any backpressure opposing inspiratory flow (auto-PEEP). Tidal volume may change abruptly with muscular activity, increased airway resistance, deteriorated lung and chest wall compliance or auto-PEEP.

In recent years, the importance to maintain consistent ventilation and nearly normal levels of $PaCO_2$ has declined. In the absence of effort, and provided that both tidal volume and end-inspiratory pause pressure ($P_{Plateau}$) are monitored, the choice of pressure or volume control makes little practical difference to physiology or outcome of the patient.

Inverse Ratio Ventilation

IVR appears not to offer any notable advantage over conventional ventilation which is applied with adequate PEEP.

Pressure Support Ventilation

During the first few days of severe ARDS, due to vigorous breathing, it may be difficult to avoid patient ventilator dys-synchrony with either volume control or pressure control. Since both require preset inspiratory times, there will be pressure limitation leading to failure to deliver full tidal volume.

Due to this, pressure support which is a flow cycled mode may be preferred during early phase of ARDS if, patient ventilator synchrony can be maintained. High level of pressure support offers response flexibility. When used alone or in combination with SIMV, PSV may minimise the timing dys-synchrony that otherwise occurs between the cycling phases of patient and machine. In any form of pressure targeted or flow regulated breathing, the ventilator's alarm should be set carefully so as to avoid over application of pressure or underventilation.

BiPAP and APRV

Recognised advantage of BiPAP and APRV in patients with ARDS is that it encourages the maintenance of an open lung units with their high pressure baselines and ensures that the spontaneous breathing pattern is preserved. No definitive evidence exists to confirm their relative advantage over well adjusted traditional approach.

High Trequency Ventilation

There is little advantage over ventilation performed conventionally with equivalent attention to the principles of lung protection.

THERAPEUTIC ADJUNCTS TO MECHANICAL VENTILATION IN ARDS

- Minimise oxygen demands
- Optimise oxygen delivery
- Recruiting maneuvers
- Prone positioning
- Improve gas exchange efficiency
  - Inhaled nitric oxide/inhaled prostacyclin
  - Tracheal gas insufflations.
- Corticosteroid and other drugs

Minimise Oxygen Demands

Limit the tissue strain by adjusting pressure driving each tidal cycle (the difference between $P_{Plateau}$ to PEEP), as well as number of high pressure cycle applied per unit time, and to be kept within acceptable limits. These requirements limit tidal volume, minute ventilation and adapt methods that reduces the requirements for ventilation and oxygenation, reduces local tissues stresses and improves the efficiency of pulmonary gas exchange, all without the need for additional ventilating pressures or higher ventilating frequency.

Optimise Oxygen Delivery

The most effective and universally applicable means for avoiding the need for high ventilator pressures is to reduce the demand for them by the patient or to use lung protective criteria. Thus avoidance of high fever, pain, anxiety, agitation and metabolic acidosis reduces oxygen and ventilation demands, as well as the need for cardiac output is an important potential cofactor for VILI. Optimising oxygen delivery by improving cardiac performance and providing adequate haemoglobin concentration also minimises the ventilatory requirements.

Improving Gas Exchange Efficiency

Methods of improving gas exchange efficiency include those directed at oxygenation (inhaled nitric oxide or inhaled prostacyclin) and those that lessen wasted ventilation tracheal gas insufflation or reduction of apparatus dead space.

Lung Protection Criteria

PEEP performs its recruitment action primarily by preventing the deflation and collapse of alveoli which are opened by tidal breath. In determining optimal PEEP, two basic approaches exist that use either.

1. Mechanical criteria
    - Pressure volume curves
    - Quasistatic technique
2. Gas exchange criteria-PEEP titration curve.

Mechanical Criteria

It involves assessments that attempt to ensure that PEEP recruits "recruitable" alveoli but does not overdistend alveoli recruited. Two approaches have been reported to identify recruitable alveoli.

Pressure volume curves (Fig. 40.8): Pressure volume curve is used to set the PEEP/V_T combination between the upper and lower inflection point.

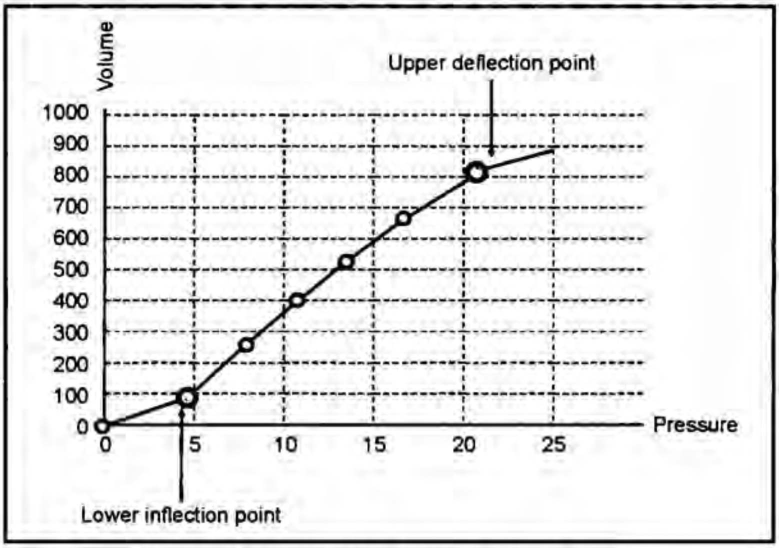

Figure 40.8: Inspiratory pressure volume curve

Thus, it is clear that inspiratory pressure volume curve of the total respiratory system identifies the minimum level of PEEP (lower inflection point) required preventing at least partial derecruitment, although a higher level may be required in many patients. Use step increase in PEEP to determine the PEEP level that gives the best compliance.

Quasistatic technique - low flow rate

This is modification of the conventional static approach which uses a very slow inspiratory flow and then measures upper and lower inflection points from the resulting dynamic pressure volume curves. The resistive element is eliminated by using a very low flow rate < 10 litre/min.

Gas Exchange Criteria—PEEP Titration Curve

Gas exchange criteria are to guide PEEP application, which involves several potential strategies.

1. PEEP titration curve to determine the lowest FiO_2 that can be achieved.
2. To use algorithms designed to provide adequate values for PaO_2 while minimising FiO_2. Constructing a PEEP/FiO_2, algorithms is usually an empirical exercise in balancing SaO_2 with FiO_2 and depends on clinician's perception of the relative "toxicities" of high thoracic pressures, high FiO_2, and low SaO_2.

In general, a commonly used "operational" range for PEEP in parenchymal lung injury is 8-25 cmH_2O. However, some argue that at least in the initial phases of lung injury, the range should be higher (e.g. 12-25 cmH_2O) and that volume recruitment manoeuvre should be performed to ensure optimal recruitment. With this approach, the patient would be weaned from PEEP only when the FiO_2 is 0.4 or less.

PEEP/FIO_2 ALGORITHM USED IN THE NATIONAL INSTITUTE OF HEALTH/ARDS NETWORK MECHANICAL VENTILATION ROTOCOL

Use this if patient's PEEP and FiO_2 are not compatible with the protocol scale and the $PaO_2 < 55$ or $SpO_2 < 88$ %. Find the box that corresponds to the current PEEP/FiO_2 settings. Make the change in PEEP or FiO_2 as indicated in the box. The approved protocol PEEP/FiO_2 combinations are indicated by ****.

	FiO_2 0.3	*FiO_2 0.4*	*FiO_2 0.5*	*FiO_2 0.6*	*FiO_2 0.7*	*FiO_2 0.8*	*FiO_2 0.9*	*FiO_2 0.10*
PEEP 5	*****	*****	↑PEEP	↑PEEP	↑PEEP	↑PEEP	↑PEEP	↑PEEP
PEEP 8	↑ FiO_2	*****	*****	↑PEEP	↑PEEP	↑PEEP	↑PEEP	↑PEEP
PEEP10	↑ FiO_2	↑ FiO_2	*****	*****	*****	↑PEEP	↑PEEP	↑PEEP
PEEP12	↑ FiO_2	↑ FiO_2	↑ FiO_2	↑ FiO_2	*****	↑PEEP	↑PEEP	↑PEEP
PEEP14	↑ FiO_2	↑ FiO_2	↑ FiO_2	↑ FiO_2	*****	*****	*****	↑PEEP
PEEP16	↑ FiO_2	↑ FiO_2	↑ FiO_2	↑ FiO_2	↑ FiO_2	↑ FiO_2	*****	↑PEEP
PEEP18	↑ FiO_2	↑ FiO_2	↑ FiO_2	↑ FiO_2	↑ FiO_2	↑ FiO_2	*****	*****
PEEP20	↑ FiO_2	↑ FiO_2	↑ FiO_2	↑ FiO_2	↑ FiO_2	↑ FiO_2	↑ FiO_2	*****
PEEP20-24	↑ FiO_2	↑ FiO_2	↑ FiO_2	↑ FiO_2	↑ FiO_2	↑ FiO_2	↑ FiO_2	**** Max R_x

Preventing Iatrogenic Injury — Ventilatory Parameter

Avoid Large Tidal Volume

There are now considerable studies indicating that the large tidal volumes used during traditional mechanical ventilation (10 to 15 ml/kg) can damage the lungs.

The pathologic changes in ARDS are not distributed uniformly throughout the lungs. Rather, there are regions of lung infiltration interspersed with regions where the lung architecture is normal. These normal lung regions (which may make up only 30% of the lung) receive most of the delivered tidal volume. Large tidal volume results in overdistension of normal lung regions, which leads to alveolar rupture, surfactant depletion, and disruption of the alveolar-capillary interface.

FiO_2 should be kept at 50% or lower

The fractional concentration of inspired oxygen (FiO_2) should be kept at 50% or lower to minimise the risk of oxygen toxicity. Arterial oxygen saturation (SaO_2) should be monitored instead of arterial PaO_2 because SaO_2 determines the oxygen content in arterial blood. A SaO_2 above 90% should be sufficient to maintain oxygen delivery to peripheral tissues. FiO_2 to be reduced below 60%, with help of external PEEP.

Other Measures which Help Outcome

Reducing Lung Water

The measures that are considered for reducing lung water are diuretics and PEEP. Unfortunately, neither measure is likely to be effective in ARDS.

Diuretics

Diuretic therapy can reduce lung water by increasing colloid osmotic pressure (increased plasma protein concentration) and decreasing capillary hydrostatic pressure.

- *Not very useful in inflammatory oedema:*
  Although diuretics play significant role in watery hydrostatic oedema, the situation is different in ARDS. The lung infiltration in ARDS is an inflammatory process and diuretics do not reduce inflammation. Considering the pathology of ARDS, routine use of diuretics is not advocated.
- *More useful when renal water excretion is impaired:*
  When renal water excretion is impaired, the use of diuretics to minimise or reduce fluid overload seems more appropriate. The best way to prevent fluid overload is to maintain an adequate cardiac output. Whenever diuretics are used, haemodynamic monitoring must

be used to make sure that the diuresis is not adversely affecting the cardiac output.

Positive end-expiratory pressure:

The use of PEEP was popularised in ARDS because of the presumption that lung water could be reduced by this maneuver. However, as demonstrated the application of PEEP does not reduce extravascular lung water in ARDS, nor does the prophylactic use of PEEP reduce the incidence of ARDS in high-risk patients. In fact, high levels of PEEP can actually increase lung water.

The effects of graded administration of PEEP on lung water in non-cardiogenic pulmonary oedema are increase in lung water. The increase in lung water may be the result of alveolar overdistension mentioned earlier, or may be the result of PEEP-induced impairment of lymphatic drainage from the lungs. Thus, the bulk of evidence indicates that PEEP is not a therapy for ARDS to reduce lung water. Instead, PEEP is a measure that helps to reduce iatrogenic lung injury by allowing ventilation with low inflation volumes, and by allowing like FiO_2 to be reduced to less toxic levels.

Important Points About Baby Lung

- Volume control should possibly only be used for very short postoperative periods.
- Set tidal volume or inspiratory pressure level to give a tidal volume that is corrected for the size of "the baby lung", i.e. 5-7ml/kg of ideal body weight.
- Decrease tidal volume and if necessary, accept permissive hypercapnia. Monitor effective compliance closely.
- If the effective compliance is 20 ml/cmH_2O and should be 80, the ventilated lung is one quarter the size of normal, e.g. "body weight 20 instead of 80".
- Always use an adequate PEEP, which will usually be not lower than 7 to 10 cmH_2O. The hypoxaemia is the most dangerous part-not the hypercapnia. Keep pH in arterial blood above 7.20.

Specific Therapies

The following therapeutic measures are aimed at reversing the pathologic lung injury in ARDS.

Steroids

High-dose steroids have been evaluated for their ability to reduce the inflammatory lung injury in ARDS. Unfortunately, the results do not favor the use of steroids in ARDS, at least not in the early stages of the illness. The following is a brief summary of the available studies.

High-dose Methylprednisolone

1. *Within 24 hours:*
   High-dose methylprednisolone (30 mg/kg intravenously every 6 hours for 4 doses) given to patients within 24 hours of the diagnosis of ARDS has not improved outcome or reduced mortality. In fact, one study showed a higher mortality associated with steroid therapy in ARDS.
2. *Prophylaxis:*
   High-dose methylprednisolone (same dose as above) given as prophylaxis to patients with sepsis syndrome did not reduce the incidence of ARDS.
3. *Late duration-2 weeks duration:*
   Low-dose methylprednisolone (2 to 3 mg/kg/day) given to 25 patients with late ARDS (2 weeks duration) who had evidence of active fibrinoproliferation (leading to irreversible pulmonary fibrosis) resulted in a beneficial response in 21 patients and an 86% survival in the responders.
4. *Complication:*
   Secondary infections are increased in patients receiving high dose methylprednisolone for ARDS.

Surfactant

Aerosolized surfactant has proven effective in improving outcomes in the neonatal form of respiratory distress syndrome, but it has not met with similar success in adults with ARDS.

Antioxidants—Nitric Oxide

Oxygen metabolites play an important role in neutrophil-mediated tissue injury and neutrophil mediated tissue injury may play an important role in the pathogenesis of ARDS. That is why there is considerable interest in the possible role of antioxidants as a specific therapy for ARDS. Nitric oxide can improve oxygenation and reduce pulmonary artery pressures in ARDS, because inhaled nitric oxide (5-20 ppm) reduces pulmonary hypertension and intrapulmonary shunting. A two year long study in adults with ARDS showed no improvement in survival, mortality was unchanged and there is concern regarding possible increase in oxidant mediated injury.

Warning Sign of Worsening the Patient

Warning Signs for Change of Ventilator Strategy

- Decrease in oxygenation and/or increase in peak airway pressure.
- Change in compliance and simultaneous change in dynamic characteristics.

Cardiovascular Warning Signs

- Drop in perfusion pressure to vital organs.
- Extrapulmonary reasons for decrease in oxygenation.

Anti-MODS Warning Signs

- Vital organ function deteriorating, e.g. brain, kidney. GIT tract, lung

Outcome of Parenchymal Respiratory Failure

Depending on the numbers of other organs involved, mortality rates can approach 100%. Under these circumstances. Conversely, lung injury from lung only processes may be associated with a mortality rate of less than 30%. Initial oxygenation impairment is not a good predictor of mortality in ARDS, although persistence of lung injury is. Survival of patients with ARDS actually may be improving and it is reflection of better overall ICU management rather than any new single modality.

CHAPTER

41

Ventilatory Strategy for Head/Brain Injury

Brain injury requiring mechanical ventilatory support may be due to trauma, tumours or cerebral oedema. To decide ventilatory strategy in above conditions one should know effects of artificial ventilation on intracranial pressure and the cerebral circulation.

CEREBRAL AUTO-REGULATION

Cerebral auto-regulation (within the range of mean arterial pressures of about 60-150 mmHg) protects cerebral circulation from major swings in systemic arterial pressure and ensures that cerebral blood flow remains constant independent of the perfusion pressure. The cerebral perfusion pressure (CPP) is the difference between mean arterial pressure (MAP) and intracranial pressure (ICP) and is the principal determinant of cerebral blood flow. When cerebral auto-regulation fails, rise in intracranial pressure decreases cerebral perfusion pressure reducing cerebral blood flow.

Mean arterial pressure and intracranial pressure are most important constituents, which can be therapeutically manipulated, thus related to the cerebral perfusion pressure in the following way:

CPP = MAP – ICP

Normal value of CPP: 60-90 mmHg

Effects of Positive Pressure Ventilation

Positive pressure ventilation increases intracranial pressure by reducing venous return from the head, thereby decreasing cerebral perfusion pressure. These effects are exaggerated in patients with cerebral tumours, head injuries, though effect is not marked in patients with normal intracranial compliance.

Cerebral Perfusion Pressure and Changes in $PaCO_2$

The cerebral perfusion pressure is affected by changes in $PaCO_2$, since cerebral blood flow and $PaCO_2$ is roughly linear. Hypocapnia (decrease in $PaCO_2$) leads to a contraction of the brain vessels and in turn reduces the blood flow. Hypercapnia (increase in $PaCO_2$) leading to a dilation of the brain vessels, increases the blood flow. These changes in $PaCO_2$ can be controlled by altering ventilation. Therefore, ventilation (control of $PaCO_2$) influences the cerebral circulation which changes intracranial pressure. A decrease of cerebral blood flow by about 4% can be anticipated by every 1 mmHg of acute reduction in $PaCO_2$.

Effects of Hypercapnia on Normal and Damaged Brain Vessels

In damaged brain tissue, particularly in oedematous areas, there is a disturbance of auto-regulation with persistent vasodilation and vasoparalysis. These changes are caused by cerebral lactic acidosis as a result of increased anaerobic metabolism.

The effect of $PaCO_2$ is mainly on undamaged or normal blood vessels comparative to vasoparalytic vessels of damaged brain. This mechanism depends on effect of $PaCO_2$ on arterial bed and type of artery. Hypercapnic acidosis induces cerebral pre-capillary arteriolar vasodilation, which depend on acidosis rather than CO_2. Though ATP sensitive potassium channels play a major role, calcium activated potassium channels are also responsible for this phenomenon.

Steal Response

Cerebral acidosis (hypercapnia) produces vasodilatation of undamaged vessels supplying the normal brain thereby increasing the cerebral blood flow and the blood volume of the normal brain. This effect must be considered carefully in a patient with risk for raised intracranial pressure. Steal phenomenon which is associated with hypercapnia, diverts the blood away from the damaged areas to the normal brain producing hypovolaemic zones around contusions. However, it increases the intravascular volume of normal brain by enhancing blood flow through it thereby damaging the normal brain. At the same time, a higher interstitial pressure is transmitted to the damaged areas exacerbating the ischaemia in a diseased brain.

Effects of hypocapnia on brain vessels

Hypocapnia results in alkalosis, and the leftward shift of the oxyhaemoglobin dissociation curve impairs oxygen release from the haemoglobin to the cerebral tissues.

Inverse Steal Phenomenon (Robin Hood Phenomenon)

The cerebral vasoconstriction that hypocapnia induces can worsen cerebral ischaemia and produce lactic acidosis. Hypocapnia leading to an increase in circulation in the oedematous area is called inverse steal phenomenon. This paradoxical effect occurs as result of vasoconstriction in undamaged brain with consequent reduction in intracranial volume and intracranial pressure of normal brain. However, there is a redistribution of blood flow whereby blood flows from the undamaged brain into the traumatised vasoparalytic areas.

RECENT ADVANCES IN THERAPEUTIC HYPERVENTILATION

The effect of hyperventilation is therefore a combination of several factors:

- Reduction in intracranial pressure caused by vasoconstriction
- Inverse steal phenomenon
- Compensation for interstitial lactic acidosis through respiratory alkalosis.

CO_2 is a potent vasodilator and an acute reduction in arterial $PaCO_2$ results in cerebral vasoconstriction, a reduction in cerebral blood volume and a resultant decrease in intracranial pressure. This was the basis of using controlled hyperventilation as a basic element in the treatment of cerebral oedema/head injury, as an effective, rapid means of reducing ICP in selected patients such as head injury and cerebral oedema.

Such a strategy has proven harmful and no longer recommended. It is unknown whether the use of short periods of hyperventilation to sudden lowering of intracranial pressure is harmful or not. Because of more recent concerns about cerebral ischaemia following hyperventilation, more emphasis has been placed on the maintenance of cerebral perfusion pressure than on simple ICP manipulations.

Arterial Pressure Oriented Approach to Maintain CPP

Therapeutic hyperventilation, using ICP only as an end-point, produces a local inverse steal response with a paradoxical increase in cerebral blood flow. This promotes swelling of the contused brain which then compresses the undamaged normal brain tissue surrounding the ischaemic zone.

Recent work suggests that a reduction in arterial pressure is worse for patients as compared to an increase in ICP. Therefore, an arterial pressure oriented approach to CPP rather than an intracranial pressure oriented approach is recommended. It is worth noting that most treatments that reduce ICP are a consequence of cerebrovascular vasoconstriction. This will have deleterious effects upon regional oxygen and substrate delivery. The therapeutic goals recommended are to maintain a mean arterial pressure of at least 90 mmHg and a CPP of 70 mmHg. To achieve this, pre-load optimisation and vasopressors are sometimes required.

GOALS OF VENTILATORY STRATEGY IN HEAD/BRAIN INJURY

Early therapeutic intervention in patients with severe head injury is directed towards:

- Airway control
- Avoiding hypercapnia
- Maintaining cerebral oxygenation
- Control intracranial pressure (ICP).

INDICATIONS FOR INTUBATION AND MECHANICAL VENTILATION IN HEAD/BRAIN INJURY

Patients with severe head injury require tracheal intubation and assisted ventilation intubation is only tolerated with muscle relaxation and sedation unless patient is deeply comatosed.

- *Repeated seizures*
- *Coma causing inadequate protection of the airway:*

  Gastric aspiration may occur, which compromises both airway and gas exchange. Patients with diminished levels of consciousness may be unable to protect their airways and may require intubation for this reason alone. Gas exchange abnormalities are common with severe head injuries due to alteration in ventilation to perfusion ratios, increase in physiological intrapulmonary shunt and accumulation in extra vascular lung water. Aspiration of gastric contents not only involves risk of asphyxia but also gives rise to acid pneumonitis and aspiration pneumonia thereby increasing the risk in a moribund patient.
- *Neurogenic pulmonary oedema:*

  Neurogenic pulmonary oedema is mediated by sympathetic reflexes, can cause alveolar filling and further worsen gas exchange. Clinically evident pulmonary oedema, occurs less frequently in non lethal head injuries, but may be associated with significant decreases in lung compliance.

- *Glasgow coma score:*
  The more profound decrease in the levels of consciousness is often associated with increase in ICP and is indications for control of ventilation. Routine practice is to intubate patients with a Glasgow coma score < 8.
- *Abnormal blood gases*
  - PaO_2 with oxygen mask less than 75 mmHg
  - $PaCO_2$ greater than 45 mmHg or less than 32 mmHg
- *Mean intracranial pressure higher than 15 mmHg*

GENERAL PRINCIPLES OF MANAGEMENT

Preference of Oral Intubation

Many head injured patients are intubated via the nasal route because of ease and convenience as well as the concern of cervical injury. But, it is ideal to use oral ETT in this patient to avoid purulent sinus infection and to avoid high incidence of pulmonary infection due to bacterial reservoirs whenever there is no cervical spine injury.

Avoid Excessive Hyperventilation

Caution must be taken to avoid excessive hyperventilation.

Maintain Oxygenation

Hypotension and hypoxia may exacerbate pre-existing cerebral injury and worsen the outcome. It is crucial therefore, that head injured patients must be kept well oxygenated.

Control of Intracranial Pressure

The control of intracranial pressure may work at cross purposes with efforts to clear secretions and maintain adequate gas exchange. Unfortunately, methods used to help clear secretions, e.g. postural drainage or tracheal suctioning may produce marked increases in ICP and are often inadvisable.

Methods used to control intracranial pressure usually involve bed elevation, adjusted hyperventilation, manitol administration, ventriculostomy drainage and paralytic agents. Patients with elevated ICP are frequently positioned with the head end of bed elevated and immobilised for prolonged periods.

VENTILATORY STRATEGIES IN BRAIN/HEAD INJURY PATIENT

Mode of Ventilation

Volume controlled ventilatory modes are preferred in head injured patients since they can deliver constant volume of gases in the presence of changing lung mechanics. Pressure controlled ventilatory modes are contraindicated because the minute volume, and therefore $PaCO_2$ can vary unless the patient is closely monitored. PSV combined with SIMV can be used as a weaning mode for such patients. During mechanical ventilation, paralysis is used when sedation alone is inadequate to control the patients their ICP.

Volume controlled ventilation is most efficient because effects are consistent, no significant detrimental effects are seen when applied to normal lung and they help to prevent atelectasis and hypoxaemia due to constant tidal volume.

FiO_2

The initial fraction of inspired oxygen should be set at 1, and then decreased by 10 to 20% until a safe level (below 0.6) is attainted. It is recommended to wait for least 20-30 minutes between changes in FiO_2 to allow the arterial PaO_2 to reach a new steady state level. Initial increase in FiO_2 will help to improve oxygenation, however to increase PEEP and change the I:E ratio (to improve oxygenation), this should be done under ICP monitoring.

Tidal Volume

Earlier all mechanically ventilated patients were ventilated with moderate tidal volumes as a traditional criteria and trying to maintain normocapnia. This is no longer followed in all cases of mechanically ventilated patients except patients with brain injury and relatively normal lungs. In head injury, a tight $PaCO_2$ control is required to avoid undesirable episodes of brain ischaemia and hyperaemia.

In a patient with normal compliance, the use of larger tidal volume of 15-18 ml/kg with corresponding reductions in ventilatory rate may be useful in reducing mean airway pressures. This technique should not be used however in patients with decreased chest and lung compliance. This technique increases peak airway pressures and is potential for producing high volume high pressure lung injury.

Minute Ventilation

Minute ventilation (tidal volume and respiratory rate) should be adjusted to keep the $PaCO_2$ level between 33-37 mmHg. Hyperventilation probably has its most deleterious effect during the first 24 hours after severe head injury, when cerebral blood flow is typically at its lowest. It is therefore recommended, that $PaCO_2$ can be maintained at approximately 35 mmHg within the first 24 hours after injury. After 24 hours $PaCO_2$ can be maintained in the range of 30 to 35 mmHg if ICP cannot be controlled.

If hyperventilation ($PaCO_2 < 30$ mmHg) is essential (i.e. when CSF drainage, elective ventilation with paralysis and sedation, and mannitol have failed to control ICP) then careful and continuous monitoring of the arterial pressure, the cerebral perfusion pressure and the arterial and venous blood gases should be done.

PEEP

Ventilator setting that acts to increase mean airway pressure may exacerbate ICP by impending venous return to the chest. The application of PEEP, CPAP may cause increase in ICP. Although the addition of end expiratory pressure, by impending venous return, may cause increases in ICP in some patients, this effect is not observed in others. Mild increase in ICP may be compensated for by corresponding increase in mean arterial pressure to maintain cerebral perfusion pressure in patients requiring the additional PEEP for oxygenation.

PEEP should be limited to <5 mmHg unless required for oxygen exchange. Hypoxaemia due to reduced lung volume is common after head injury with or without chest X-ray changes and if uncorrected, it may persist for several days. The addition of a low level of PEEP increases lung volume, decreases venous admixture, increases arterial PO_2 and allows the FiO_2 to be lowered to less toxic levels.

Brain injury patient with normal lung compliance, PEEP may increase intracranial pressure (ICP) due to impedance of venous return from the head. However, with non compliant lungs, transmission of excessive pressure generated by PEEP is minimal due to dampening effect of lung parenchyma and it does not cause as much adverse effect on a patient's ICP. The effect of PEEP on the ICP is variable with increases in ICP more common in the presence of low cerebral compliance.

The intracranial effect of PEEP can be blunted or eliminated by positioning head-injured patients at a 15 to 30 degree head-up position or limiting PEEP to 10 cm. H_2O. The treatment of hypoxia is given first priority even if there is rise in ICP.

Precaution at the Time of Termination of PEEP

Termination of PEEP should be performed in a stepwise fashion. Careful monitoring of hemodynamic parameters and the arterial blood gases over 1 to 2 days is necessary. This will lessen the chance of recollapse of recruited alveoli. Abrupt discontinuation of PEEP can lead to a reflex hypertension. Hypertension will give rise to engorgement of cerebral tissues with defective autoregulation with subsequent elevation of ICP.

Supportive Measures

To ensure an adequate cerebral perfusion pressure of 55 mmHg, vasopressors (Noradrenalin) are sometimes required. Renal compensatory mechanisms and changes in pH limit the effectiveness of hyperventilation after 24 hours. $PaCO_2$ values should not fall below normal to avoid cerebral hypoperfusion.

High Frequency Ventilation is Harmful

Although HFV eliminates the normal cyclic variations of the ICP present in positive pressure ventilation, most head injured patient with low intracerebral compliance do not permit optimal utilisation of this technique because of a concurrent pulmonary pathology. When the HFV settings are adjusted to achieve the desired arterial PaO_2 and $PaCO_2$ levels, an effectively high PEEP in the pulmonary circuit results and raise the ICP. Currently, HFV has no place in the routine management of head injured patient.

WEANING FROM SEVERE HEAD INJURY

- Normalisation of $PaCO_2$ by reduction in ventilatory support
- Complete spontaneous ventilation on T peace or CPAP
- Careful assessment of airway reflexes prior to extubation
- Patient in vegetative state requires tracheostomy

PATIENT WITH ABNORMAL VENTILATORY PATTERN

Patients with brain injury may have abnormal ventilatory patterns and varying degree of autonomic instability. The abnormal pattern may be also due to pulmonary infection or cardiac failure, which will reverse if cause is treated. If abnormal pattern remains in spite of treating the cause it means it is central in origin. In spite of abnormal pattern, mechanical ventilation should be withdrawn after careful monitoring.

Patient should be observed for deterioration by clinical examination and monitoring of blood gases for acute ventilatory failure (increased $PaCO_2$ and decreased pH). If blood gases are maintained for a reasonable period of time (24-48 hours), then it is suggestive that the abnormal pattern is central in origin and mechanical ventilatory support is not necessary. Ventilatory pattern frequently returns to normal after several months following brain injury.

	Ventilatory Strategy
Mode or A/C mode	Volume controlled ventilation (minimal flow ventilation)
FiO_2	Depending on the patient
Inspiratory flow	60 to 70 liters/min
Tidal Volume	10 to 12 ml/Kg
Respiratory rate	12 to 14 breaths/min
I:E ratio	1:2 (avoid IRV)
PEEP	5 to 7 cm H_2O.

Since compensation for hypocapnic cerebral vasoconstriction occurs fairly quickly, deliberate hyperventilation can at best be considered a temporary measure for the urgent control of intracranial hyperventilation until more definitive measures for the control of the latter are taken. Compensatory mechanisms then gradually restore the pH in the local milieu towards normal over the next few hours, cerebral blood flow also slowly normalises. If at a subsequent point of time, an attempt is made to normalise the systemic $PaCO_2$ by reducing the level of hyperventilation, a rebound increase in intracranial pressure can occur, cerebral hyperemia can result in reperfusion injury or cerebral haemorrhage in previously ischaemic regions of the brain.

In cases of neurologic injury, with normal intracranial pressure, prophylactic hyperventilation is certainly not desirable. Hyperventilating the patient with normal intracranial pressure might reduce the option of hyperventilating the patient at a later stage should the intracranial pressure subsequently rise. Thus for example, should the head trauma patient (who is prophylactically hyperventilated) subsequently develop a rise in intracranial pressure, further hyperventilation would be required to bring already low CO_2 down to extremely low levels in order to decrease the intracranial pressure.

Furthermore, by increasing minute volume in an already hyperventilated patient, the intrathoracic pressure would be expected to increase and so impede the venous return, including that from the cerebral veins, further increasing intracranial pressure and compounding the problem. Therefore it is probably better to avoid prophylactic hyperventilation in patients with neurological injury, so that this modality is available as an option for decreasing the intracranial pressure should an elevation in the ICP occur at a subsequent stage.

CHAPTER

42

Ventilatory Strategy for Neuromuscular Diseases

The most important and primary muscle of respiration is the diaphragm, a flat dome shaped muscle attached to the lower ribs. Diaphragm is innervated by the third through fifth cranial nerves. Contraction of external intercostal muscles raises the ribs. It also provides stability to the rib cage and prevents its inward collapse during diaphragmatic contraction.

NEUROMUSCULAR DISEASE ASSOCIATED WITH RESPIRATORY IMPAIRMENT OR FAILURE

1. Spinal cord injury
2. Motor neuron disease
    - Poliomyelitis
3. Peripheral neuropathies
    - Autoimmune/inflammatory—Guillain/Barre
    - Shellfish poisoning
    - Acute intermittent porphyria
4. Neuromuscular junction
    - Myasthenia gravis
    - Botulism
5. Muscle disease
    - Endocrine myopathies—hyperthyroidism, hypothyroidism.

In neuromuscular disease, ventilatory failure is due to diseases that impairs transmission of neural input from the respiratory center to the respiratory muscles or due to conditions that prevent proper contraction of respiratory muscles. Typically, the respiratory center itself is unaffected and initiates a normal respiratory drive. In these patients, lungs are normal and gas exchange is normally not affected. If the patient is having type II respiratory failure with a fall in PaO_2 associated with rise in $PACO_2$ then the A-aDO_2 is normal unless significant atelectasis occurs.

ANAESTHETIC SIGNIFICANCE OF ASCENDING PARALYSIS

Reduced Lung Volumes and Capacity

With ascending paralysis such as Guillain Barre Syndrome or traumatic spinal injury (quadriplegia), above T_{10} level reduces vital capacities, other lung volumes and gives rise to distortion of rib cage with inspiratory effort. Patient with neuromuscular disease respiratory impairment is generally proportional to the number of inspiratory muscle involved. This has the following effect:

- Alveolar arterial oxygen gradient increases early during course of disease before development of hypercapnia.
- Due to decrease in lung volumes, ventilation perfusion mismatching occurs particularly in dependent portions of the lung.
- Alveolar hypoventilation, inhibited intercostal and accessory muscle activity, rapid shallow breathing inducing dead space ventilation worsens gas exchange abnormalities during the sleep. Central drive in patients with neuromuscular diseases and chest wall disorder increases despite muscle weakness inducing rapid shallow breathing. Motor neuron disease, e.g. poliomyelitis, muscle disease like muscular dystrophy can be often associated with thoracic scoliosis, often reduces respiratory compliances.
- Respiratory compliance decreases due to reduction in lung volume which adds elastic load of already weakened inspiratory muscle. Eventually the diaphragm becomes fatigued because it has to carry maximum load.

Reduction in Ability to Cough

Weakened abdominal muscles reduce the ability to cough, as reflected by decreased peak expiratory flow rates. Peak expiratory flows of less than 160 lits/min need cough assistance to prevent accumulation of airway secretions. Inspiratory muscle weakness and glottic dysfunction worsens cough impairment. Involvement of bulbar muscle impairs speech and swallowing which increases risk of aspiration and upper airway collapse.

Abnormal feedback

In patient with muscle disease (e.g. muscular dystrophies), abnormal regulation of breathing has been attributed to abnormal feedback from respiratory muscle receptors. Abnormal central ventilatory control is seen in patients with myotonic dystrophies giving rise to abnormal regulation of breathing.

Abnormal breathing during sleep

This abnormal pattern during sleep is attributed to many factors:

- Abnormalities in control of ventilation
- Respiratory muscle weakness
- Chest wall deformities
- Bulbar dysfunction
- Obesity.

INDICATION FOR MECHANICAL VENTILATION

- Subjective symptoms—orthopnea, morning headache, daytime somnolence, impaired cognitive function, e.g. memory, concentration, fatigue
- Measurement of vital capacity < 25% of predicted
- Nasal sniff pressure < 25 cmH_2O
- Recurrent acute respiratory failure episodes requiring intervention
- Changes in gas exchange indicating progressive failure.

SUBJECTIVE SYMPTOMS OF PROGRESSIVE RESPIRATORY FAILURE

Dyspnea, increased use of accessory neck muscles, paradoxical breathing, tachycardia, sweating and inability to say more than few words together are nonspecific findings of progressive respiratory failure. Severe hypercapnia and acidosis and/or hypoxaemia will produce altered mental status.

However, slower progression of respiratory impairment can present without overt signs. These patients usually may complain of sleep disturbances, somnolence during day time, headache, fatigue, or exhibit cognitive impairment associated with daytime hypercapnia with partial compensation. Those with generalised muscle weakness experience orthopnea because of combination of diaphragmatic fatigue, unstable chest wall and cephalad pressure exerted by abdominal contents on the diaphragm when lying supine. Patients with quadriplegic often prefers supine position because diaphragm assumes a greater appositional area with respect to abdominal wall, increasing the resting length and forçe generation.

MEASUREMENT OF VITAL CAPACITY AND TOTAL LUNG CAPACITY

Vital capacity < 25% of predicted.

Measurements provide a guide to the progress of respiratory muscle weakness. It is not very good indicator since 50% reduction of respiratory muscle strength occurs before the VC and TLC are reduced. Patients with less than 10% of predicted VC have tolerance of assisted ventilation.

MODE OF VENTILATION

Non-invasive ventilation is an accepted form of respiratory support in patients with neuromuscular disease and chest wall disorder. In irreversibly paralysed patients, in whom recovery of spontaneous breathing is not expected, or in patients with drug overdose, in whom spontaneous resolution of ventilatory failure can be expected within hours, mechanical ventilation in the control mode (with high tidal volumes and rapid inspiratory flow rates) to perform all the work of breathing, is an entirely acceptable option.

If pulmonary mechanism is normal high tidal volumes can be used. In these patients, mostly ventilatory drive is normal; such patients may show preference to for large tidal volume (12-15 ml/kg body weight) at high flow rates. The loss of cough reflex may result in aspiration and patients are prone for atelectasis when adequate tidal volumes are not used.

In reversibly paralysed patient like Guillain—Barre Syndrome, virtually any effective mode of mechanical ventilation (e.g. A/C, SIMV or PSV) is acceptable. The A/C mode has the advantage that patients can easily react to changing metabolic needs by changing the assist rate and adjusting minute ventilation. In any case, frequent high—volume sighs help to prevent atelectatic complications.

ROLE OF PEEP

Patients with decreased FRC, PEEP application maintains alveolar patency during expiration and reverses the ventilation perfusion mismatch that result in hypoxaemia. Neuromuscular weakness patient's are expected to have normal respiratory compliance, however, they have dependent alveolar collapse due to reduced FRC and dependent alveolar unit fails to open during inspiration. PEEP has beneficial effect in reversing dependent alveolar collapse and helps to open dependent alveolar unit during inspiration.

NON-INVASIVE VENTILATION – BIPAP

Expiratory positive airway pressure (EPAP) application of BiPAP has same role like PEEP. However in BiPAP, there must be parallel increase in the inspiratory positive air way pressure (IPAP) to provide adequate ventilation. While pressure cycled ventilation can compensate mask leaks partially, excessive leaks can prolong the inspiratory phase, and worsen intrinsic PEEP and patient ventilator asynchrony.

Patient with muscular dystrophy may be associated with cardiomyopathy. In these patients, high levels of PEEP or high EPAP and IPAP can lead to decreased cardiac output due to impaired left ventricular function. Non-invasive positive pressure support has been used with success for the long term ventilation of patients with neuromuscular injury.

CHAPTER 43

Weaning from Mechanical Ventilation

Mechanical ventilation is an effective form of therapy given to a patient with respiratory failure. Unlike heart, which itself is a pump, the lungs are incapable of moving gas volumes (breathing) and are therefore dependent on ventilatory pump to perform the work of breathing. Over 90% of the critically ill patients require mechanical ventilation to do ventilatory pump function. Patients are ventilated because there is imbalance between the amount of work required for the breathing and the capability of the ventilator pump to perform that work.

When ventilatory support is no longer required the process of transferring task from ventilator to the patient is called '***liberation***' 'discontinuation' 'withdrawal' and most commonly 'weaning'. Many physicians use the term 'weaning' to describe the process of discontinuing ventilatory support regardless of the time frame involved. The word weans means 'to withdraw'. It also means 'to tease away gradually'. This weaning approach is sometimes referred to as "taking off the ventilator" or simply "discontinuing the ventilator."

As the patient's condition improves, attention should be given to remove the ventilator as quickly as possible. Unnecessary delays may increase the complication rate for mechanical ventilation, e.g. nosocomial infection, pneumonia, airway trauma as well as the cost. However, discontinuing the ventilatory support too soon may place a burden on the patient's ventilatory capacity and cause respiratory muscle fatigue. This may delay weaning even further causing weaning failure. Weaning will fail unless the underlying illness is treated or cured. The focus of weaning is trying to correct underlying illness and not weaning process.

Every department has its own approach, own preference and prejudice regarding weaning from mechanical ventilation. It is supported by clinical evidence that weaning in 95% of the patients presents no problem. 2% of the patients can not breath because they have no lungs and only 3% of patients weaning can be difficult because something is wrong with the ventilatory pump.

STAGES OF WEANING

There are seven stages of weaning:

1. *Pre-weaning*:
   When no attempt at weaning is desirable. Every ventilated patient begin at this stage and some patients never get beyond this stage. Cardiovascular unstable patient, maintaining oxygenation with 80% oxygen and PEEP of 15 cm H_2O cannot be even considered for weaning.
2. *Suspicion:*
   First thought comes in mind of physician that the patient just might come off the ventilator successfully—diagnostic triggering.
3. *Measure predictor:*
   This is the time of measuring and interpreting weaning predictors.
4. *Weaning trials:*
   This is time decreasing ventilator support. Support is either removed abruptly and completely (T tube trial) or gradually decreased over hours or days.
5. *Extubation:*
   This is extubation of the patient who tolerated stage 4 or reinstitution of mechanical ventilation in a patient who failed the weaning trial. This is either weaning success patient or weaning failure patient.
6. *NIV post-extubation:*
   This is continued ventilator support after extubation using non-invasive ventilation. This stage is applied to very few patients.
7. *Reintubation:*
   This is usually accompanied by the reinstitution of mechanical ventilation.

MISCONCEPTION REGARDING WEANING

The practice of weaning from mechanical ventilation has been dominated by a number of misconceptions:

- Removal of endotracheal tube reduces the work of breathing.
- The longer the duration of mechanical ventilation, the more difficult is the weaning.

- The method of weaning determines the ability to wean.
- Diaphragm weakness is a common cause of failed wean attempt.
- Aggressive nutrition support improves the ability to wean.

PATHOPHYSIOLOGY OF WEANING

The common criteria of weaning are adequate gas exchange, lung volumes, respiratory muscle strength, and oxygen cost of breathing. All these criteria can never be fulfilled in all patients and there is always individual variation in ventilator dependent patient.

Volume criteria are more applicable to patients with neuromuscular weakness or postoperative patients but not to patients with intrinsic lung disease (where capillary and alveoli are not speaking with each other).

TIMING OF WEANING

The period of weaning compromises from the total ventilator support is withdrawn till the patient breathes spontaneously through his natural airway. Timing changes depending on indication of mechanical ventilation. Elective postoperative ventilation should pose neither problem nor challenge for anaesthesiologist.

PRIMARY GOALS OF WEANING

The primary goals of mechanical ventilation are to improve the function of ventilatory pump, to reduce the work of breathing and hence cardiac workload. Mechanical ventilation also provides rest to respiratory muscles which reduces the oxygen cost of breathing. The imbalance between respiratory capacity and ventilatory load should return to normal or near normal. However, single most important criteria to consider before weaning is whether there has been a significant improvement or reversal in the primary pathology of the patient that led to intubation and ventilation. The ability to tolerate the discontinuation of mechanical ventilation depends on both mechanical component of the respiratory system and its gas exchanging capacity.

INDICATIONS OF VENTILATORY SUPPORT

Indications are based on failure of two physiological mechanisms:

Acute respiratory failure (ARF) is the end result of either ventilator pump failure or gas exchange failure.

Pulmonary Ventilatory Failure — Hypercapnic Respiratory Failure-Type I

It is characterised by insufficient elimination of CO_2 giving rise to increased $PaCO_2$ (hypercarbia), which is due to reduced alveolar ventilation. In later stage decreased PaO_2 (hypoxaemia) is evident, e.g. COPD.

Pulmonary Parenchymal Failure — Hypoxaemic Respiratory Failure-Type II

It is characterised by inadequate oxygenation, due to all disorders of alveolar-capillary membrane, leading to reduced PaO_2 and an increased alveolar arterial oxygen difference ($A\text{-}aDO_2$). It has got early hypoxaemia, hyperventilation (increased minute ventilation), hypocapnia (decreased $PaCO_2$) and late stages hypercarbia, e.g. ARDS, pulmonary oedema.

Ventilation and oxygenation are two different entities and clear distinction should be made in the management. Ventilatory pump failure is to be treated with increasing alveolar ventilation and gas exchange failure is to be treated according to the underlying intrinsic disease of lung. Arterial blood gas (ABG) estimation is single most reliable tool to diagnose respiratory failure, the clinical diagnosis is only at late stage, except neuromuscular disease where you can identify acute respiratory failure clinically in advance and deterioration of blood gases means you have waited too long.

WEANING CRITERIES

Before weaning, the following clinical criteria should be considered:

- Significant improvement or reversal in the primary pathology of the patient leading to improved pulmonary mechanics and better gas exchange
- Haemodynamically stable patient
- Adequacy of normal renal function
- Good clinical condition without sepsis or CNS depression
- Normal acid base balance
- Protection of airway reflex
- Psychologically stable and mentally prepared patient
- No severe abdominal distension
- No fluid overload
- Adequate rest and sleeping time.

Determinants of weaning outcome depending on clinical criteria

Abnormalities in oxygenation or failure of ventilatory pump are responsible for prolonged mechanical ventilation. To understand why patient has failed to wean it is necessary to identify which of the above physiological variable has altered.

Haemodynamically Stable Patient Without Sepsis

The patient must be haemodynamically stable before weaning. Left ventricular dysfunction, myocardial ischaemia, cardiovascular instability and pulmonary oedema is associated with decreased weaning success. A significant number of ventilator dependent patients often have coexistent cardiovascular disease. High metabolic demands from overloaded ventilatory muscle which is associated with compromised oxygen delivery during reduction of ventilatory support, may precipitate cardiovascular failure. This phenomenon is more common with ventilator dependent COPD patient. After better control of heart failure and diuresis weaning success may improve.

A tachycardia – heart rate > 100 – 120 beats/min, or a bradycardia-heart rate < 60 – 70 min, hypotension – BP < 90/60 mmHg' or severe hypertension – >180/110 mmHg should be treated before considering discontinuation of ventilatory support.

Normal Acid-base Balance

The patients must have a normal acid-base balance before weaning and presence of abnormalities must be corrected before weaning. An increase in metabolic rate due to fever, sepsis, shivering, seizures or agitation can also increase oxygen demand and should be prevented or treated. The presence of metabolic acidosis compensates with respiratory alkalosis and places an increased load on the respiratory muscles. Metabolic alkalosis, on the other hand, may decrease ventilatory drive.

Adequacy of Renal Function

Adequate renal function is required to maintain acid-base homeostasis, electrolyte and fluid balance.

- *Metabolic acidosis:*
  Renal insufficiency can produce metabolic acidosis, which will increase ventilatory load and can affect respiratory muscle strength.
- *Electrolyte imbalances:*
  Adequate potassium, magnesium and phosphate levels are necessary for adequate ventilation. Electrolyte disorders can impair ventilatory muscle function. Mechanically ventilated patients are prone to develop electrolyte imbalances due to inadequate oral intake, excess loss (diuretic use, vomiting, and diarrhoea) or inadequate replacement (total parenteral nutrition).
- *Fluid overload:*
  Fluid overload compromises respiratory function by reducing both lung and chest wall compliance. The congestion of the lungs impairs ventilation and oxygenation.

- *Decreased metabolism of neuromuscular blocking drugs:*
  Persistent paralysis can be due to decreased metabolism of neuromuscular blocking drugs with impaired liver or renal function or use of continuous infusion.
  In patients with chronic renal failure, some form of renal replacement therapy may be necessary for successful weaning.

Other Important Elements

Nutrition

In critically ill patients, there exists an imbalance between nutritional support and metabolic demand which reduces total body mass, reducing respiratory muscle mass and muscle strength. Nutrition must be adequate to maintain respiratory muscle strength and mass. Malnutrition has significant effects on the body that could impair weaning due to respiratory muscle weakness. Electrolyte abnormalities which are common in ICU will also reduce respiratory muscle strength. Low serum albumin always correlates with many difficult wean situations.

A daily intake of 1–1.5 g/kg of protein is considered adequate. Excessive carbohydrates may increase carbon dioxide production and increase the respiratory load. The role of fats in the diet to reduce CO_2 production is still unclear. However, it is observed that overfeeding, particularly of high carbohydrate meals can compromise ventilator muscle loading. Adequate nutrition and avoidance of high carbohydrate load is paramount importance.

Infection

Prevention and control of systemic and pulmonary infection is helpful in weaning, due to its profound effects on the weaning process. Systemic metabolic effects of infection not only directly impair respiratory muscle functions but also compromise oxygen delivery. Pneumonia can increase ventilatory muscle loads and thereby compromise the weaning process. Sepsis and resultant multiple organ dysfunction syndromes are the major causes for mortality in ventilator dependent patients.

Neurologic Function

The patient must be awake and alert, free of seizures and able to follow instructions. Adequate CNS function is needed to ensure a stable ventilatory drive, adequate secretion clearance (cough and deep breathing) and protection of the airway (gag and swallowing reflex). The most common neurological problem with sedation is that it depresses consciousness which may then further increase ventilation duration.

Excessive sedation can impair cough, clearing of secretions, compromise airway defenses thereby reducing the ability to protect against aspirations. Critical care neuropathy also makes weaning difficult. Level of consciousness, anxiety, depression, dyspnoea and motivation can all have an impact on weaning success or failure.

Protection of Airway Reflex

Most weaning indices do not evaluate the airway. The patient must have the adequate gag and cough reflex. The decision to discontinue ventilatory support must be separated from the decision to extubate. Some patients can be successfully weaned from the ventilator, but not fully awake or unable to clear secretions due to the decreased levels of consciousness associated with aspiration following extubation. This type of patients should not be extubated; the tracheal tube should be left in place or in long term tracheostomy is indicated. Some patients may have an adequate gag reflex, but may need assistance with ventilation. In such patients, noninvasive ventilation can be attempted.

Psychological Factors

Subjective clinical state such as pain, fatigue, and dyspnoea must be avoided.

Various Criteria Used for Weaning Indices/Predictor of Success

Prior to extubating a patient recovering from acute respiratory failure, some basic criteria must be fulfilled. Clinical experience is most important criteria in predicting weaning outcome. A variety of parameters have been used to aid assessing the patient's capability to discontinue mechanical ventilation. The reliability to predict weaning success has been generally disappointing. The following are the parameters used:

1. Gas exchange
    - Oxygenation
    - Ventilation
2. Breathing pattern/Ventilatory mechanics
3. Neuromuscular capacity/drive – Respiratory muscle strength
4. Measures respiratory muscle load
5. Integrative indices

Gas Exchange

1. *Oxygenation criteria:*
   Weaning success will be more likely, if the patient is adequately oxygenated. Poor oxygenation is always associated with weaning failure. Inadequate oxygenation is mostly associated with abnormalities of respiratory capacity and ventilatory load which delay weaning.

FiO_2	$< 0.4 - 0.5$
PaO_2 (mmHg)	> 60 mmHg with $FiO_2 < 0.4$ and PEEP $< 5 - 7$ cm H_2O
SaO_2 (%)	> 90 %
$S\bar{V}O_2$ (%)	> 60 %
PaO_2/FiO_2 ratio	> 200 or < 350
$(A - a)\ PO_2$ gradient	< 350 mmHg at an FiO_2 of 1
Or	
PaO_2/PAO_2 ratio	> 0.35

No lactic acidosis, adequate cardiac output, blood pressure.

- *PaO_2 and SaO_2:*
  The patient should be able to maintain a PaO_2 of at least 60 mmHg on FiO_2 of < 0.4. A PaO_2 of 60 mm Hg corresponds to SaO_2 of about 90%. If pulse oximetry is used to monitor patient's oxygenation status, the pulse oximetry O_2 saturation (SpO_2) should be maintained in the 90s range for allowance of machine inaccuracies because SpO_2 reading in critical care is accurate within 2-4% of SaO_2 estimate.
- *PaO_2/FiO_2:*
  The arterial oxygen tension to inspired oxygen concentration index is a simplified method of estimating degree of intrapulmonary shunt. PaO_2 / FiO_2 of 200 mm Hg or higher is indicative of normal physiological shunt and successful weaning.

Hypoxaemia can occur for different pulmonary and non pulmonary conditions. These conditions often lead to a combination of V/Q mismatch, shunt, severe hypoventilation, dead space ventilation, reduced oxygen delivery, diffusion defect. Although mechanical ventilation will improve oxygenation, it is necessary to identify and to treat underlying cause of lung failure. In addition, cardiac output, haemoglobin and oxygen saturation should be normal ensuring normal oxygen delivery.

2. *Ventilation criteria:*
   - $PaCO_2 > 45$ mmHg
   - pH 7.35–7.45 (apart from COPD)

$PaCO_2$: The partial pressure of carbon dioxide in the arterial blood is the most reliable indicator of the patient's ventilator status. In patients with normal lung functions, the $PaCO_2$ should be within the normal range of 35–45 mm Hg and the pH should be between 7.35–7.45. However, in COPD patient's the acceptable $PaCO_2$ is slightly higher and pH slightly lower.

Breathing Pattern/Ventilatory Mechanics

Studies have proved that patients failing to wean from mechanical ventilation commonly have a rapid and shallow breathing pattern, consequence of severe respiratory dysfunction. Irregular spontaneous breathing or periods of apnoea are not good signs for weaning success. Any of the above signs are suggestive of an increased workload that may lead to ventilatory muscle fatigue and failure. Asynchronous and rapid shallow breathing patterns indicate respiratory decompensation.

- *Spontaneous respiratory rate (bpm) > 8 < 30/min:*
  A rate greater than 30 breath per minute is associated with an increased work of breathing which may not be able to sustain by the patient, leading to weaning failure.

  Clinical assessment of adequacy of ventilation must include evaluation of any sign of respiratory distress and/or irregular ventilatory pattern. Tachypnoea (respiratory rate > 30 breaths/min) is a sensitive marker for respiratory distress but can prolong intubation if it is used as an exclusive criteria. If patient is having respiratory distress, scalene muscle can be palpated during inspiration and abdominal muscle tensing is palpable during expiration. Patients should be able to alter ventilation on command. If there is an inability to alter the ventilation on command, then the patient will not be able to sustain ventilation.
- *Spontaneous tidal volume (ml/kg) > 5-10 ml/ kg:*
  It is generally accepted that the minimal spontaneous tidal volume > 5-10 ml/kg consistent with successful weaning.
- *Vital capacity (ml/kg): 10–15 ml/kg, BW or 800–1000 ml.*
  The mechanical condition of lungs can be evaluated by measuring vital capacity and spontaneous tidal volume. It assesses the single breath respiratory muscle performance. A value of vital capacity > 10 ml/kg predicts weaning success. This has not proven to be clinically useful because it is patient dependent. Poor effort and inability to follow commands leads to wrong value. But VC <10 ml/kg will require intubation and ventilation in patients with neuromuscular diseases.
- $V_T / V_C > 0.4$

- *Minute ventilation on ventilator (lit/min) < 10 lit/min:*
  Work of breathing rises as ventilatory requirements increase. In normal patients, minute ventilation up to 6 lit is normal, however, weaning success is associated with minute ventilation (either spontaneous or assisted) less than 10 lit/min (assuming $PaCO_2$ is normal).

A high minute ventilation requirement (>10 lit) needed to normalise $PaCO_2$ which means the work of spontaneous breathing will be excessive. The patient is unlikely to be able to sustain the increased work of breathing once ventilator rate begins to reduce. An excessive minute volume requirement may result from increased carbon dioxide production.

Causes of increase in increased carbon dioxide production

- Increased metabolic rate, e.g. fever, overfeeding with carbohydrate supplements.
- Increased alveolar dead space, e.g. hyperinflation of lung, underperfused pulmonary perfusion (i.e. pulmonary embolism, decreased cardiac output.)
- Metabolic acidosis

Neuromuscular Capacity/Drive-respiratory Muscle Strength/ Pulmonary Reserve

The capacity of the respiratory muscles to perform work against mechanical and metabolic loads depends on intact neuromuscular function. Normal neuromuscular function may not predict success, if there is excessive respiratory load.

- *PI_{max} /MIP maximum inspiratory pressure:* Normal < – 20 to – 30 cm H_2O (negative inspiratory force).

  MIP does not reflect the ventilatory load but assess respiratory muscle strength which is an important determinant of weaning success. It is the amount of negative pressure that is generated in 20 to 25 seconds when inspired against occluded medical device (negative pressure manometer). The strength of the diaphragm and other muscles of inspiration can be evaluated by having the patient exhale to residual lung volume and then inhale as forcefully against closed valve. The airway is occluded for 20-25 seconds with a one way valve that allows the patient to exhale, but not inhale. The airway pressure generated by this manoeuvre is called the maximum inspiratory pressure and is used to test respiratory muscle strength. However, this requires patient effort and difficult to perform in critically ill intubated patient.

  PI_{max} is useful to identify the patients with potentially treatable respiratory muscle weakness because it tests respiratory muscle strength and weaning will be likely successful if the patient can generate PI $_{max}$ of at least–20–30 cm H_2O (negative pressure). The value less than – 20 cm H_2O was useful in predicting weaning failure, associated with CO_2 retention in patients with weak muscles.

- *Airway occlusion pressure – $P_{0.1}$ -3-4 cm H_2O:*

  Airway occlusion pressure is the pressure generated during the first 100 msec. of an occluded airway by inspiratory effort. $P_{0.1}$ is a measure of respiratory effort/breathing drive during spontaneous breathing. It has been used to assess neuromuscular output from medullary respiratory centre. It can only be carried out in the assisted spontaneous breathing/spontaneous ventilation mode.

  Measurement of Occlusion Pressure

  The ventilator keeps the inspiratory valve closed after expiration (a pressure – 0.5 mbar is taken as the beginning of inspiratory effort) and measures the airway pressure generated by inspiratory effort. The 100 msec. time interval starts when a negative pressure of –0.5 mbar (P1) is measured as a result of the inspiratory effort. A second pressure value (P2) is measured after 100 ms. simultaneously, the inspiratory valve is opened, and the patient may breath normally again. The difference between these two pressures P2-P1 is the occlusion pressure, i.e. P $_{0.1}$: (Fig. 43.1).

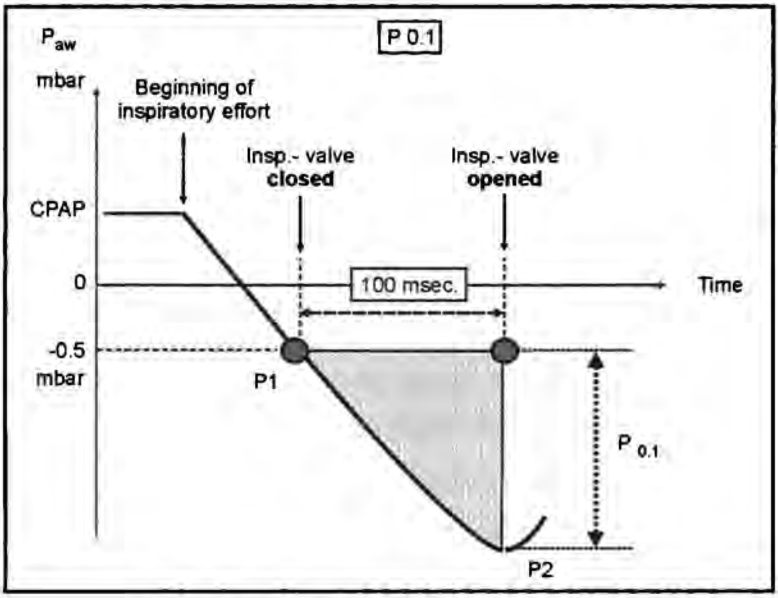

Figure 43.1: Measurement of occlusion pressure

High $P_{0.1}$

Abnormally elevated drive during spontaneous breathing could result from mismatch of mechanical load and neuromuscular function. High $P_{0.1}$ suggests considerable respiratory effort, which can only be maintained for a limited period of time. $P_{0.1}$ values for 6 cm H_2O, e.g. in patients with COPD may be an indicator of imminent ventilatory failure. Murciano examined the relationship between $P_{0.1}$ and clinical outcome in patients with COPD in acute respiratory failure. $P_{0.1}$ was markedly elevated with those individuals requiring continued ventilator support, but fell significantly in-patient's who were weaned.

Fernandez demonstrated that sensitivity and specificity in differentiating between patients who needed full or no further ventilator support were improved by using the ratio of $P_{0.1}$ to PI_{max} rather than $P_{0.1}$ alone.

Measures Respiratory Muscle Load

The respiratory muscle load should be considered as tidal volume, compliance, resistance.

Higher the load the poorer is weaning outcome. Normal load can precipitate ventilatory failure in patients with abnormal neuromuscular function. That is why respiratory muscle loads must be interpreted in relation to respiratory muscle strength.

- *Minute ventilation on ventilator < 10 lit/min:*
  Minute ventilation is the major determinant of the amount of pressure work that must be performed per unit of time and it monitors ventilatory status. If respiratory mechanics are constant, the work of breathing rises as ventilatory requirement rises.
- *Respiratory system compliance*
- *Airway resistance:*
  A very high airways resistance > 20 cm $H_2O/1/sec.$ is likely to cause weaning failure even with normal respiratory muscles due to the greater the work of breathing.

Integrative Indexes – Combined Weaning Indices

f/V_T	< 105
$P_{0.1}/PI_{max}$	< 0.15
$P_{0.1}/RR/V_T$	< 450

- *Rapid, Shallow Breathing Index(RSBI) - $f/V_T < 105$ cycles/litre*

  Rapid, shallow breathing index (f/V_T) is the ratio of spontaneous breathing frequency (cycles-breaths/min) to tidal volume (in litres) and is a good indicator of weaning success in terms of sensitivity and specificity and helps in predicting weaning outcome other than conventional weaning criteria in many patients requiring mechanical ventilation.

  Failure of weaning may be related to the development of a spontaneous breathing pattern that is rapid (high respiratory rate) and shallow (low tidal volume). Rapid and shallow breathing is common in patients who fail to wean from mechanical ventilation, because this pattern induces dead space ventilation. This ratio if normally is less than 50 cycles/litre and is often above 100 cycles/litre in patients, suggests potential weaning failure.

  To measure the f/V_T ratio, the patient is taken off the ventilator and is allowed to breathe spontaneously for three minutes or until stable breathing pattern then measure parameters. However, f/V_T is less useful in predicting weaning success in patients requiring ventilation for more than 8 days and in the elderly.

- *Oxygen cost of breathing:*

  The oxygen cost of breathing can be estimated by subtracting total body oxygen consumption during controlled ventilation from that measured during unassisted breathing. Since patients cannot perform significant respiratory muscle work during controlled ventilation, the assumption on which calculation of the oxygen cost of breathing is based probably invalid and remains unproven value.

  In normal adults, the act of quiet breathing consumes only 5% of the total oxygen consumption. When minute ventilation increases above 10 lit/min, the oxygen cost of breathing increases. The oxygen cost of breathing is a measure of the 'efficacy of breathing' or the relationship between the work performed and the energy needed to perform the work.

- *CROP – Compliance, Rate, Oxygenation, Pressure Index*

  CROP is not practical to be used in the clinical setting as it requires measurement of four variables.

Preparation of Patient Recovering from Respiratory Failure

The patient should be well rested and must be allowed to sleep at night. The level of ventilatory support must be increased for the night if required. Ensure good nutrition before starting weaning. Minimise the work of breathing by treating infection, bronchospasm and airway oedema and use pressure support ventilation. Suctioning and adequate humidification must be done to clear secretions. There is no specific formula to determine when a patient no longer needs mechanical ventilation, because weaning is a art.

TECHNIQUE OF WEANING

When the underlying problem is corrected, the patient should wean gradually regardless of the method used. There is no technique of weaning which is superior than other. If there are no contraindication regarding spontaneous breathing in terms of the underlying disease, sedation can be reduced slowly in order to increases the contribution of spontaneous breathing to the ventilation without having to change the mode of ventilation.

All ventilator parameters and inspired oxygen concentration need to be reduced before discontinuing mechanical ventilation. Strengthening of respiratory muscles slowly over period of time, helps to wean gradually, before spontaneous respiration is tolerated. One of the commonest cause of weaning failure is increased work of breathing such as a narrow airway (ETT/ tracheostomy), with additional load imposed by circuit and demand valve.

- Normalisation of the I:E ratio
- Reduce analgesic and sedation
- Reduction of the PEEP
- Reduction of the inspired O_2 concentration–FiO_2 of less than 0.5 is desirable.
- Shift to ventilatory modes which allow spontaneous respiratory effort, e.g. SIMV, PSV, BIPAP, CPAP.
- If the patient develops tachypnoea under reduced sedation, increase the depth of sedation and not necessary to switch to a "controlled" ventilation mode.

METHODS OF DISCONTINUING MECHANICAL VENTILATION

Broadly weaning method can be divided into two and each is analogous to an electrical switch. One is abrupt like off-on switch, while other likes a rheostat, i.e. gradual method.

T Piece Method—Spontaneous Breathing Trial (SBT)

Formal assessments for ability to maintain respiration must be done during spontaneous breathing rather than the patient receiving substantial ventilatory support. An initial brief period of spontaneous breathing can be used to assess patient tolerance during a spontaneous breathing trial (SBT).

This is often done using simple T-piece system, which is attached to endotracheal tube. The T-piece should have a diameter of 22 mm in all parts, with a central suction port. Ayres T-piece should not be used. Humidified oxygen enriched gas mixture at constant flow is delivered to the inlet arm of the T-piece. When the patient takes a breath, the gas is drawn into the lungs from the inlet only and patient can expire through other end.

When T-tube system is used, the gas flow to inspiratory limb must be a minimum of twice the patients spontaneous minute ventilation to meet the patient's peak inspiratory flow rate. To prevent entrainment of room air extension tubing of 12 inches long is added to expiratory limb.

Abrupt T-piece Wean Method

Abrupt discontinuation depends largely on the duration of ventilation. If patient is receiving ventilatory support for short periods, e.g. postoperative patients, he can normally be weaned and extubated without prolonged weaning trials.

Stable patients, who have been on a ventilator not more than 2-3 days, can be rapidly weaned off the ventilator once the precipitating cause is reversed. Typically, these patients have stable blood gases, fully conscious, capable of maintaining their ventilation and haemodynamically stable. This patient can be disconnected from ventilator and placed on a T-tube for a short period. If the patient is stable, arterial blood gases are maintained and then the patient may be extubated.

Clear data does not make available for optimal duration of T-tube trial. In general, it is agreed that if patient is not in distress and is comfortable or that there is absence of deterioration of gas exchange for 2-4 hours, patient can be safely extubated.

Intermittent T-piece Method—Spontaneous Breathing Trial (SBT)

Once the decision to wean is taken, the ventilator is disconnected and the patient is administered oxygen through a T-piece connected to the endotracheal tube/tracheostomy. Weaning is attempted by using spontaneous breathing trials of increasing duration, mixed with period of rest using positive pressure ventilation. The frequency and duration spontaneous breathing trials should be increased so patient can breathe without ventilatory support.

The optimal duration and frequency of these trials varies from patient to patient. The optimal duration and interval between spontaneous breathing needed to maximise the 'training effect' is unknown. There can be gradual approach of starting trial depending on duration of stay on the ventilator.

- The T-piece trial period may be 5 minutes to half an hour initially
- Patient to be placed on mechanical ventilation for ventilatory support for 1-4 hours for the rest.
- The period off the ventilator is gradually increased for upto 2 hours depending on patient tolerance.
- The tolerance of SBTs lasting 30-120 minutes should prompt for the consideration of permanent ventilator discontinuance, if blood gas and vital are stable.

There are varieties of protocol for intermittent T-Tube trials. Each user uses specified period for spontaneous breathing and ventilatory support (2 hours off, 2 hours on). Some users keep the patient off the ventilator as long as he/she tolerates. If spontaneous breathing is tolerated for 24 hours, the patient can be extubated.

One should remember that endotracheal tube also has got significant resistance. The patient may do better after extubation if the patient has ability to cough and clear secretion. It is also important to monitor the patient closely and ensure that the airway is patent at all times. Due attention must be given to tracheal toilet as necessary. It is important to ensure that the patient does not go into respiratory distress while on T-piece trials due to blockage of the tube, since it may delay weaning further.

Advantages of T-tube-trial

- The T-tube trials test both the strength and the endurance of the respiratory muscles.
- The patient feels immense satisfaction of being withdrawn from the machine and as the performance improves over the days.
- Feeding solids by mouth is possible.
- Deflation of the cuff is possible in few patients. It will decrease the airway resistance and the patient also will able to phonate and communicate.
- WOB is the least in this weaning mode, as dead space added is minimal and air way resistance depends only on length and internal diameter of tracheostomy tube or endotracheal tube, since circuit has no demand or expiratory valve.

Disadvantages of T-tube trial

- Overenthusiastic T-piece trial can result in severe respiratory acidosis and cardiac complications.
- It needs close observation and monitoring, compared to other modes, due to non-availibilty of backup.
- It does provide neither sufficient muscular activity nor sufficient rest.

T-tube trial is terminated

- Increase in respiratory rate by > 10 breath/min or to > 40 breaths/min.
- Increase or decrease in blood pressure by approximately 20 mmHg
- Increase or decrease in heart rate by approximately 20 beats/min.
- $SaO_2 < 90\%$ or $PaO_2 < 60$ mmHg
- Increase in $PaCO_2 > 45$ mmHg or decrease in pH to < 7.3
- Development of cardiac arrhythmias
- Sign of increased work of breathing
  - Accessory muscle use
  - Nasal flaring
  - Intercostal retraction
  - Paradoxical breathing
- Complaints of dyspnoea/fatigue

DETERIORATION OF BLOOD GASES IS A LATE INDICATION

Gradual Methods—Support Modes for Weaning

These various weaning modes reduce the need to alternate the patient on T-tube and ventilatory support.

- Synchronised mandatory ventilation—SIMV with PSV
- Continuous Positive Airway Pressure—CPAP
- Biphasic positive airway pressure—BIPAP
- Pressure Support Ventilation—PSV
- Volume Support—VS and Volume Assured Pressure Support—VAPS
- Mandatory minute ventilation—MMV
- Adaptive Support ventilation—ASV
- Non-Invasive Positive Pressure Ventilation—NIPPV.

Synchronized Mandatory Ventilation–SIMV

During SIMV, the patient receives fixed number of positive pressure volume/pressure cycled breath per minute and can breathe spontaneously in between these breaths. There are two ways of weaning a patient off SIMV.

- *Start patient with full support than reduce gradually:*
  SIMV can also be used to provide full ventilatory support. When SIMV rate is 12 or above it is full support. The full ventilatory support is continued till there is significant improvement in the patient's disease state.
- *Start patient with partial support:*
  Partial ventilatory support is provided to the patient right from the beginning, since patient is having sufficient spontaneous activity. Partial ventilatory support encourages patient's effort and minimizes patient-ventilator asynchrony. However, if a patient has respiratory muscle fatigue, change to provide full ventilatory support to unload respiratory muscles.

  The level of support is reduced gradually only after the clinical improvement is confirmed. By reducing the SIMV rate, it is possible to produces graded increase in inspiratory effort performed by the patient.

SIMV with PSV

Most clinicians now use a combination of SIMV and PSV during weaning as initial ventilatory support.

- Set SIMV rate 2 breaths/min lower than AC mode or 80% of AC mode rate + PSV 5-15 cm H_2O + PEEP – 7-9 cm H_2O.
- A rhythmic respiratory pattern is important
- Reduce the SIMV rate in steps – 1-3 breaths/min.
- Monitor the patient clinically and do the arterial blood analysis after 30 minutes
- If pH remains above 7.30 or 7.35, reduce the rate further – 1-3 breaths/min + Reduce the PEEP in steps of 1-2 cm H_2O until PEEP is 3 to 5 cm H_2O.
- If deterioration develops, increases the SIMV rate.
- Allow the patients respiratory muscle to rest at night by increasing the SIMV rate and/or increasing the PSV.
- When SIMV rate becomes 3-5 breaths/min., stop SIMV and continue PSV 5-15 cm H_2O + CPAP– 7-9 cm H_2O.
- Reduce the PSV in steps of 2 cmH_2O until PSV is 5 to 6 cm H_2O.

If the patient is requiring only 5 cm H_2O PSV + CPAP about 6-8 cm H_2O, and uses less than 40% oxygen, he can be disconnected from the ventilator or even extubated. The 5 cm H_2O PSV is said to be required to overcome the work of breathing imposed by the ventilatory tubings, endotracheal tube etc. Hence, the PSV level need not be reduced beyond 5 cm H_2O. This concept is controversial.

It is observed that the ventilatory load on the patient during SIMV weaning occurs in the early phase but during PSV weaning, it occurs much later. It is important for the clinician to be alert early during SIMV weaning and late periods during PSV weaning.

Continuous Positive Airway Pressure (CPAP)

CPAP can be used instead of T-tube trials. CPAP maintains lung volumes by preventing airway collapse. A severely diminished FRC and reduced lung compliance greatly increases the alveolar opening pressure. Since PEEP increases the FRC, pulmonary impairment may be prevented or improved by early application of PEEP therapy.

The normal or physiological PEEP applied by the vocal cords during breathing is bypassed by the endotracheal tube or tracheostomy. If the patient is breathing spontaneously, decreased lung compliance always increases the work of breathing and if severe enough can lead to fatigue of the respiratory muscles and ventilatory failure. This may encourage alveolar collapse during T-piece trials. The use of CPAP is said to avoid this atelectasis during weaning, because CPAP maintains lung volumes by preventing airway collapse. In addition, the ventilator serves as a monitor during weaning because of the alarms on the ventilator. In case of COPD, CPAP/PEEP plays role to reduce inspiratory effort rather than gas exchange.

BIPAP

The transition from controlled ventilation, via the weaning phase, to complete spontaneous breathing is achieved by approximating the two pressure levels P_{high} and P_{low} and by increasing the time T_{low}, and thereby reducing the ventilatory frequency.

Pressure Support Ventilation (PSV)

In general, pressure targeted modes of ventilation with their variable flows are more effective in providing patient ventilatory flow synchrony though volume assisted breaths are also effective provided careful adjustment of flow rates are done. CPAP can be also used with pressure support ventilation.

Depending on level of pressure support minute ventilation increases, which reduces work and oxygen cost of breathing. This helps for gradual transition from assisted ventilation to spontaneous breathing. Respiratory frequency reduces due to unloading of respiratory muscle which is very useful guide in weaning as to how effective this unloading is.

The pressure support level is initially set up so that the ventilator does the entire work of breathing and the level can be gradually reduced as the patient recovers. Since the patient determines the rate, the inspiratory time and the flow, it is a more comfortable mode of ventilation during weaning. However, it is necessary for the patient to have a reasonable respiratory rate to trigger the ventilator. If the patient goes into apnoea, no machine breaths will be delivered unless the ventilator has an inbuilt back-up mechanism. In addition, PSV does not guarantee normal tidal volumes. Very small tidal volumes may lead to atelectasis.

This mode can be used as stand mode alone. It is advisable to provide full ventilatory support at night to allow the patient to rest regardless of the weaning approach.

- Start pressure support level at 5-15 cm H_2O, increased gradually if needed.
- Pressure support can be increased until a desired spontaneous rate is achieved, 25 breaths/minute or less. This has got clinical relevance since increased spontaneous tidal volume corresponds with a decreased spontaneous rate.
- The pressure support level gradually decreased by 3-6 cm H_2O increment if patient tolerates the weaning process.
- Minimum pressure support 5 - 8 cm H_2O level is necessary to overcome airway resistance offered by ETT and circuit.

  $$P_{min} = PIFR \times R$$

  P_{min}-Minimum pressure support– enough pressure to overcome the resistance of the ventilator circuit without augmenting the patient's spontaneous inflation volumes.

  PEFR–Peak inspiratory flow rate during spontaneous breathing (measured while the patient breaths spontaneously through the ventilatory circuit).

  R – Resistance to inspiratory flow during mechanical ventilation

  $R = P_{peak} - P_{plateau} / V_{inspi}$ (inspiratory flow delivered by ventilator)

  The minimum pressure derived in this fashion varies from patient to patient and to be continued for period of spontaneous breathing and is continued until the patient is extubated.
- Extubation can be considered when the patient's blood gases and vital are stable.

Excessive level of pressure support, especially in patients with obstructive lung disease can further trap air, making it difficult for patients to trigger the ventilator. This may give false impression that the breathing frequency has slowed, when in fact it is the ventilator failing to recognise patient efforts.

Volume support (VS) and volume assured pressure support (VAPS)

Volume support is a mode very similar to pressure support and the inspiration is triggered by the patient. The ventilator adjusts the pressure support on the basis of the previous breath to ensure that the patient gets the preset tidal volume. Volume assured pressure support is a mode that entails adjustment of pressure support to get the preset tidal volume in the same breath. Each breath is analysed and the necessary adjustment made in the same breath itself. Thus, every breath is maintained at the desired tidal volume.

Mandatory Minute Ventilation (MMV)

MMV is used to wean patients off the ventilator. The physician chooses the optimum minute volume required by the patient. The ventilator analyses the first few breaths of the patient and computes whether the patient can reach the target minute volume. If there is a shortfall, the required minute volume is given to the patient by using mandatory breaths as required. The number of mandatory breaths delivered by the ventilator is reduced as the patient improves and contributes more to the minute volume.

Some ventilators augment the patient's minute volume to the desired level by altering the level of pressure support (PSV). As the level of pressure support is increased, the patient receives larger breaths to maintain minute volume. The support is automatically reduced as the patient recovers.

One of the drawbacks of MMV is that the patient can "cheat" the ventilator by taking shallow, rapid breaths thus reaching the required minute volume. This however, is inefficient as the tidal volume approaches more and more, the dead space and the alveolar ventilation can drastically fall. If used carefully, MMV can be a useful mode of weaning.

Adaptive Support Ventilation (ASV)

This is variation of MMV. MMV has got risk and limitation of rapid shallow breathing, inadvertent PEEP and excessive dead space ventilation. ASV was designed to minimise these risks. In ASV, preset MMV is maintained independent of the patient's activity. Based on the age and weight of the patient, microprocessor will calculate the minute volume of the patient.

The physician selects the percentage of the minute volume to be delivered by the ventilator. It can be set at 100% initially and gradually reduced to lower levels. The ventilator using pressure support primarily does the augmentation of minute volume. Once the patient is capable of achieving 80% of his minute volume, extubation can be considered after a brief period of T-tube trial. ASV is believed to reduce the need of readjustments at time of weaning.

Non-invasive Positive Pressure Ventilation (NIPPV)

NIPPV is reasonable adjunct to treatment in recently extubated patients who are at risk for deterioration and may have possible need for further mechanical ventilatory support.

The most commonly used form of non-invasive ventilation is Bi-level positive airway pressure (BIPAP).

Best Method

There are no universally acceptable regimens to recommend that one method is better than the other. Different patients may respond differently to different techniques.

The best approach is the one the physician is most familiar. The method chosen should include a careful assessment of the patient's condition, which should be optimised before weaning. The important factor in successful weaning is not the method used, but the ability to recognize when a patient is capable of spontaneous unassisted breathing. Most patients require ventilator assistance because lungs do not work and weaning does not improve lung function. When lung function is adequate to support spontaneous breathing, the patient will wean, regardless of the weaning technique.

However, randomised controlled trials have reported the poorest weaning outcomes with SIMV. Two prospective, randomised, controlled trials reported that two thirds of patients are successfully extubated after the first T-piece trial. In those who failed the first-day T-piece trial, the pooled results of these studies show no difference in the outcome (duration of ventilation) for T-piece and PSV. Both T-piece and PSV were superior to SIMV in both studies.

There is no evidence to support the use of new ventilatory modes to facilitate weaning. The pooled results from two prospective, randomised, controlled trials suggests reductions in the duration of mechanical ventilation, the length of ICU stay, mortality and the incidence of nosocomial pneumonia with extubation to NIPPV was used as a weaning technique.

The role of ventilatory muscle conditioning is not clear. Continuous low levels of ventilatory work as in pressure support ventilation can help develop endurance conditioning. Maximal ventilatory effort, even for short periods as in T-tube trials can help develop strength conditioning.

Monitoring

The Patient

The patient should be observed for any sign of respiratory distress. It is vital that the patient does not tire himself during weaning. Weaning could be delayed with diaphragmatic fatigue. The patient's respiratory rate, tidal volume, oxygen saturation and if available, the end-tidal carbon dioxide provides valuable clue to the clinician. The patient needs to be closely and continuously monitored, especially if on a T-piece trial.

Ventilation

If the patient is not doing well, an arterial blood gas analysis should be done before continuing with the weaning. Arterial carbon dioxide is the single best index of ventilation and has to verify that the patient is doing well before extubation too.

Oxygenation

The pulse oximeter is a sensitive indicator of oxygenation status of the patient.

If any doubt exists about the validity of the pulse oximeter readings, an arterial blood gas analysis should be done. However, it is important to realise that a significant drop in alveolar ventilation may be missed if the patient is receiving high inspired concentration of oxygen and the pulse oximeter is used as the sole monitor.

Cardiovascular Status

The patient's heart rate, rhythm, and blood pressure should be monitored continuously. Any change in rhythm or bradycardia, tachycardia should be monitored. Silent myocardial ischaemia may occur during weaning.

Extubation

Weaning indices helps to predict whether the patient can breathe for himself without a ventilator. Weaning and extubation are two separate decisions. Those who will be successfully extubated will have the resolution of the disease state that led them to the ICU. The decision to extubate should be based on the assessment of the patient meeting weaning criteria as well as an assessment of the airway patency and protection.

- Haemodynamic stability
- Airway pressure must be reduced to minimum
- Absence of sepsis

- Adequate oxygenation status
  - $PaO_2 > 75$ mmHg with an $FIO_2 < 40\%$
  - Low PEEP/CPAP
- Adequate ventilatory status.

IMPORTANT POINT TO REMEMBER

Clear fluids may be given up to four hours prior to extubation. Enteral feeding is stopped six hours prior to the proposed time of extubation.

A physician capable of reintubating the patient should be available before extubation and equipment necessary for intubation should be checked and kept ready.

Extubation should be postponed if patient has got myocardial ischaemia or an upper GI bleed or a procedure that requires reintubation.

Role of Steroids to Prevent Postextubation Complications

There is no proven benefit of corticosteroids to prevent postextubation complications.

Cuff-Leak Test

Sore throat and hoarseness are common after extubation. The 'cuff-leak test' can be used to check airway obstruction due to laryngeal oedema after extubation. After thorough oropharyngeal suction, and aspiration of nasogastric tube, the endotracheal tube cuff is deflated and the proximal end of the endotracheal tube is blocked transiently. And if the patient is unable to breathe around the tube, laryngeal oedema should be suspected.

Postextubation stridor can be mild, moderate or severe. Mild or moderate stridor may be treated with nebulisation. Aerosolised epinephrine (2.5 ml of 1% epinephrine or 2.25% racemic epinephrine in 3 ml saline) or dexamethasone (1 mg in 4 ml saline) has proved effective in reducing postextubation laryngeal oedema. If severe, the patient may require reintubation till the oedema settles down. Inspiratory stridor is a sign of severe obstruction (more than 80%).

Since the glottic sensations are blunted for 4-8 hours after extubation in any patient intubated for more than 8 hours, enteral feeding should be withheld for 4-6 hours after extubation.

Tracheostomy

Tracheostomy is commonly performed for critically ill, ventilator dependent patients to provide long-term airway access. The benefits include improved patient comfort, more effective airway suctioning, better oral hygiene, ability to eat orally and a more secure airway. These may be expected to reduce the incidence of ventilator complications, ventilator-associated pneumonia and accelerated weaning from mechanical ventilation.

The optimal time for performing tracheostomy is a controversial issue. Decision becomes simpler if straightforward and consistent approach is used. There is an increased risk for tracheal stenosis associated with repeated tracheostomy incisions. If possible tracheostomy should not be performed in patients who have had prior tracheostomy until a waiting period of more than one week has passed. Though the best way of long term airway management is tracheostomy, it does not prevent microaspiration around the tracheostomy cuff because of capillary action. However, it helps to prevents gastric and oral secretion and massive aspiration of food particles.

Tracheostomy should be considered after an initial period of stabilisation on the ventilator when it becomes apparent that the patient will require prolonged ventilator assistance. Based on general recommendations of the consensus conference on artificial airways, if the pathology has persisted for 7-10 days and is not predicted to resolve within the next 3 days, it is best to proceed with tracheostomy. Tracheostomy then, should be performed when the patient appears likely to gain one or more of the benefits mentioned above. The beneficial effects of early tracheostomy are in reducing both days in intensive care unit and on the ventilator. It will also avoid laryngeal injury and accidental loss of airway. If the patient shows clinical improvement, it is best to wait and permit extubation.

Patients who are expected to benefit are the following:

- Those requiring high levels of sedation to tolerate translaryngeal tubes
- Those with marginal respiratory mechanics who may benefit from the lower airway resistance of a tracheostomy
- Those who may derive psychological benefit from being able to speak and eat.

Failed Extubation

The most common cause of failure to wean is an imbalance between ventilatory capability and ventilatory demand. Most often, there is a tendency to miscalculate the capacity of the patient's ability to sustain adequate ventilation leading to too soon a wean. In such situations, there is likely to be some pathological process that needs further treatment. Other reasons cited are myocardial ischaemia, critical illness poluneuropathy, unsuspected neuromuscular transmission, psychological dependence, poor oxygenation or cardiovascular instability.

Chronic Ventilator-dependent Patients

In spite of all advances, a small number of patients may remain dependent on a ventilator. Such patients may have an irreversible pathological process leading to ventilatory failure. They may need long term rehabilitators care, if such units exist. Wheel-chair mounted ventilators are available to give them as much mobility as possible. All attempts should be made to reduce the extent of support required by them. Chest physiotherapy, suction and other respiratory care would help to keep the patient as comfortable as possible.

The Problem Wean/Weaning Failure

Rapid Breathing

It is necessary to distinguish anxiety from respiratory fatigue or cardiopulmonary insufficiency, since it gives rise to rapid breathing. Anxiety is accompanied by hyperventilation, where tidal volume is usually increased, whereas pathologic causes of wean failure causes rapid shallow breathing. Therefore, increase in tidal volume with unchanged or decreased $PaCO_2$ suggest anxiety, whereas decrease in tidal volume with unchanged or increased $PaCO_2$ suggest true wean failure.

Abdominal Paradox

The respiratory movements of the abdomen can provide information about functional integrity about diaphragm. During spontaneous respiration diaphragm contracts, and descending abdomen are increasing intra-abdominal pressure which pushes anterior wall of abdomen outwards when chest expands.

When the diaphragm is weak, the negative intrathoracic pressure by accessory muscle of respiration pulls diaphragm upwards into the thorax. This decreases the intraabdominal pressure and causes paradoxical inward displacement of the abdomen during inspiration. This is called abdominal paradox, during weaning it is sign of diaphragmatic weakness, but it is reliable only during quite breathing.

During labored breathing, contraction of accessory muscle of inspiration can overcome the contractive force of diaphragm and pull diaphragm into thorax giving rise to abdominal paradox. Quite breathing is uncommon during weaning it may not be sigh of weakness but sign of labored breathing and resume ventilatory support.

Conclusion

The most important criterion in determining whether a patient is ready for ventilator discontinuance or weaning is significant improvement or reversal of the disease state or condition that caused the patient to be placed on the ventilator in the first place. Clinical judgment can be complemented by the use of several weaning criteria. No one weaning method has been proved superior to another. Close monitoring of the patient is mandatory. The decision to extubate must also take into account the patient's ability to maintain the patency of the airway. Weaning failure is usually due to the respiratory workload exceeding the patient's respiratory capacity. The goal in long-term ventilator dependent patients would be to reduce the amount of support, reduce the invasiveness of support and increasing the patient's level of independent function.

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Shaila Shodhan Kamat MBBS DA MD (Anaesthesiology)

Dramatics, cookery, embroidery, tailoring, reading, spirituality, gardening... are just some of the areas that interestShaila and she has excelled in all these fields over the years. She completed MD (Anaesthesiology) in 1983 from Goa Medical College, University of Mumbai after having successfully completed her MBBS from the same college.

Accolades

"Dr Shaila Kamat is an experienced teacher in Anaesthesiology leading to mechanical ventilation. Her students have acclaimed her abilities to simplify difficult concepts. Her presentations on any topic are well organized indicating her understanding of the material."

—Vijay Deshpande, Assistant Professor (EMERITUS)
Georgia State University Atlanta, Georgia

"Rarely do you come across a student who remembers you as a teacher—Dr Shaila is one of those rare breeds. Right from the beginning she displayed a very sincere and hard working character coupled with a very active mind. Her scientific curiosity and perseverance to excel at any given task are the sole attributes which have taken her to great heights and made her the master of both the art and the science of Anaesthesia. Her extreme compassion and gentleness towards the patients leave you refreshed!!"

—Bharati Sardesai, Consultant Anaesthesiologist, Panjim, Goa, India

"I have known Dr Shaila Kamat for the past 4 years. She has a magnetic personality. She is very sound academically and takes special efforts to promote her students in all the conferences and in making publications. Her special interest lies in the art of living and she has given some wonderful lectures on that."

—Meenu Chadha, Chief Anaesthetist, OT Superintendent and Pain Consultant
Vishesh Hospital and Diagnostic Solutions, Indore (Madhya Pradesh), India

"Dr Shaila Kamat is truly a very gentle and pious lady, always enthusiastic to help her students and colleagues. In simple words, Anaesthesia is defined as the science and art of putting patients to sleep for surgery. Science is well documented in books. It is the effective delivery which is indeed artistic and makes the difference in a patient and surgeon's satisfaction and this is not mentioned in any literature. This is where Dr Shaila Kamat scores over rest of the community. The way she anaesthetizes patients is an art which all of us should pick up from her. She is an excellent teacher and always ready to impart knowledge and experience to her residents. Her command over the subject results in students gaining knowledge about Anaesthesia and related fields effortlessly."

— Harbhajan Saini, Chief Anaesthetist and Intensivist
Deepak Hospital, Ludhiana, Punjab, India

"A wonderful person Dr Shaila is! An immensely committed and sincere teacher! Always striving to live up to this noble profession! She is a fair and just taskmaster, and a stickler for punctuality and details. Though she appears a stern disciplinarian outwardly, it is only when one gets to know her heart that one finds that she has so much affection to give to all. She has always maintained that knowing one's basics thoroughly and keeping oneself updated is the only way to supplement one's years of experience in the field, and this ideal she follows, no matter what hardships she has to face, considering her huge appetite for books on Anaesthesiology."

—Anita Naik, ESIH Consultant, Margao, Goa, India

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